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Archives of Neurology and Psychiatry

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CONDITIONED REFLEXES *

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Reflexes are considered mostly as purely mechanical actions. It is supposed that from an entirely physicochemical point of view one can understand them more easily than one can actions of will or of purpose. In his classic book on the "Integrative Action of the Nervous System," Sherrington, however, emphasized not only that the simple reflex is a purely abstract conception, but that the reflex also is an action the initiation of which is traceable to events in the environment, and he began his analysis of reflexes with an instance which every one would consider a real action: An animal turns its head in response to sudden illumination. He added that in the light of the darwinian theory, every reflex must be purposive and that the effect of every reflex is to enable the organism in some particular respect better to dominate the environment. Sherrington took the reflex as the unit of nervous function. Even such a—I should like to say psychologic—reflex conception as Sherrington's, however, cannot state completely the actions of an animal; Graham Brown stressed the rhythmic autonomous functions of the nervous centers.[1] If one considers the reflex as purposeless and purely mechanical, one will never pass from the reflex to action. An action is not composed of reflexes, but makes use of the reflexes. One can consider the reflex as the basis of action only if one sees in the reflex an action with a special purpose.

Sherrington did not deny that reflex actions might be connected with a psychic tendency, but he expressed the belief that the only methodical way of investigation is that of studying objective reactions. Pavlov expressed a similar point of view, but he went farther than Sherrington in that he tried to study complicated behavior reactions without, however, caring what data introspection might yield. Introspection is considered by him a wholly unreliable method of investigation. Pavlov [2] developed his ideas in connection with extremely interesting facts—the

* Submitted for publication, Jan. 2, 1929.

* From the Phipps Clinic, Johns Hopkins Hospital, Baltimore, Dr. Adolf Meyer, Director.

1. Compare also Weizsaecker's point of view. Reference in Schilder: Medizinische Psychologie, Berlin, Julius Springer, 1924, pp. 374.

2. For the complete literature on conditioned reflexes, see Beritoff, J.: Ueber die individuell-erworbene Tätigkeit des Zentralnervensystems, J. f. Psychol. u. Neurol. **33**:113, 1927.

conditioned reflexes. "These reflexes were studied on the salivary glands of dogs. When a savory morsel was presented to the nose of the dog saliva was secreted, and if on repeated occasions a bell was sounded at the same moment that the food was presented, the sound of the bell, after a certain number of repetitions of this conjunction of impressions, sufficed to evoke a flow of saliva."[3] The question should naturally arise whether this admirable experiment does not show quite the same principle as the so-called associations. Why should it not be possible and obligatory to study also the psychic facts? Bechterew, who studied the combination of defense reactions after the electric stimulation of the sole of the foot with indifferent stimuli, spoke, indeed, of associated reflexes and thought that introspection reveals valuable facts. Nevertheless, he believed that objective study is far superior to introspection. Neither Bechterew nor Pavlov is interested in what is going on psychologically when a conditioned reflex occurs. Bechterew mentioned only that in the moment of the conditioned movement the irritation of the sole is mentally revived. Pavlov and Bechterew try to interpret social behavior and the higher activities of man along the lines of their investigations. Do they not forget that in their basic material, in every single experiment they make, there is already an implication of the psychic life, and that the facts of psychic life cannot be interpreted by experiments in which the psychic life is included but neglected as an unanalyzed factor? "Every external agency which acts on receptor organs can become an individual signal for the provocation of a special movement or secretion. It is only necessary that this external agency work several times in the act of secretion or movement, or that it precedes and follows it quickly," according to Beritoff. The objective formulation cannot deceive us. Among these facts there are also psychologic facts of true objective actuality.

It is a great mistake—the behaviorist's mistake—to believe that an exclusively objective terminology for psychic facts, borrowed from nonpsychic experience, necessarily gives a better approach to the facts. It is true that anger, for instance, implies some movements of the muscles, changes in the blood pressure and the secretions, etc. Every emotion is doubtless connected with a specific set of changes in the organism. But one understands the attitude of the person much better when one says, "He is angry," than when one gathers all those facts together which stand meaningless until one finally sees clearly, "That is anger"! The observation of psychic facts is not secondary to objective observations. Furthermore, one observes these psychic facts directly in others as well as in oneself. He cannot separate and single out the psychic life from the whole of the psychobiologic integration, which, according to Adolf Meyer, includes body as well as mind. In the description which

3. Formulated by McDougall: Outline of Abnormal Psychology, New York, Charles Scribner's Sons, 1926, pp. 571.

Watson [4] gave of children he made use of the immediate perception of mentally integrated activity. But he avoided seeing these same facts in himself. Watson, with his point of view, is greatly interested in the objective aspect of the conditioned reflexes; he merely neglects the subjective factor in them.

There is, however, another point that needs consideration. The salivary reflex is by no means a constant reflex. Pupils of Pavlov have mentioned its great variability. Also there is no doubt that conditioned reflexes have not as close and limited a relation to the structure of the segmental nervous apparatus as have the primary reflexes. They belong in some way to another order of facts. I may mention only that the conditioned reflex disappears rather quickly when not "conditioned" again. Pavlov and Bechterew have carefully studied the influences which inhibit the conditioned reflex. One must realize that the term reflex covers phenomena which belong to different levels of integration of the nervous system. The knee jerk, for instance, belongs to a rather primitive level. It is true that even the knee jerk is not constant and invariable—K. Goldstein pointed that out especially—but the degree of constancy and invariability is high in comparison with the salivary responses. Of course a phenomenon like the knee jerk offers greater possibilities for a purely physicochemical interpretation than any reflex of a higher integrative level. Most people whose knee jerk is provoked feel that there is something going on which has nothing to do with their personality. "It is involuntary." "I did not intend it," they say.[5] But one is not justified in identifying the knee jerk with the salivary reflex, and much less so with the conditioned reflex. Those who, with Goldscheider, want to have the neuroses studied on a conditioned reflex basis, overlook the fact that the term reflex is used in very different ways, and do not see that the psychic mechanisms are included in the conditioned reflex they want to study. Neither can I agree with those who believe that the study of the so-called conditioned reflexes can help one understand the neuroses better than does psychobiologic formulation. In the conditioned reflex and in the psychologic facts, either from our own introspection or from the introspection of others, the same mechanisms come out. One gets a deeper insight by introspection. It is true that introspection should be controlled by every possible method, and the simpler methods of conditioning reflexes help in this control. Therein

4. Watson: Psychology from the Standpoint of a Behaviorist.

5. My own observation does not agree with the answers of my subjects. I feel that the knee jerk also belongs in some way to a field in which the ego has an influence. And I think that a careful psychologic study would reveal that one has at least the impression that one allows the knee jerk to take place and that it is felt as an "indirect activity" of the individual (Die Neurosenfrage, Deutsche med. Wchnschr., 1927, nos. 29 and 30.)

lies their value. But one must know what a conditioned reflex really is, and here introspection should not be neglected. I therefore undertook the task of studying the psychic processes going on when a conditioned reflex is built up.

EXPERIMENTAL OBSERVATIONS

My procedure was as follows: The subject sits comfortably on a chair, his right hand resting on two electrodes so that the palm touches one and the fingers the other. Before the subject is an electric light. Five seconds after the light flashes[6] the subject receives a painful electric shock, strong enough to make him withdraw his hand. This procedure was repeated from fifteen to twenty times at one sitting, six days each week. After the second week, the stimulus sometimes came late and sometimes dropped out, but not more than once at a sitting. Usually the subjects were allowed to believe that some technical error had occurred. They were asked to report all their thoughts and feelings during the experiment, especially during the time between the flash of light and the electric shock. From choice, I have not made use of a recording system, but have contented myself with the observation of the movements of the subjects. The experiments took place in a quiet room containing only a few objects. The tests were made on five subjects.

Subject 1.—During the first experiment, on October 26, the subject, Dr. P., aged 30, felt expectation; and during the second one, relaxation. During the sixth experiment, he "wants to get away from that." During the seventh experiment, there was a feeling of tenseness; during the eighth, he recorded thinking about his wife, children and shopping. In the expectation period of all the subsequent experiments, many thoughts concerning the day's life went on. The appearance of the light provoked some thoughts about an electric lamp at home.

On October 27, he remarked, "The discomfort consists more of an expectation than of actual pain." During the sixth experiment he told about a tenseness which came immediately before the shock. Very often, associations with events of the daily life came in.

On October 29, he said that his attention was centered on the thought that something disagreeable was going to happen. In the following days, the tenseness diminished.

On November 1, in the twentieth experiment, no shock was given. He did not make any movement and felt a little unpleasantness and disappointment when the shock did not come.

On the next day, the shock came late during the nineteenth experiment. He felt a little disappointed.

On November 3, he remarked that his anticipation and tenseness had almost completely disappeared. The interfering thoughts which he had at the beginning also dropped out.

On November 8, the shock was given late. He experienced a feeling of disappointment when the shock failed to come. In the following days, he spoke of some feelings of expectation.

On November 12, during the twelfth experiment, he had, with the flash of light, the feeling that the stimulus should come now and he felt disappointed when it did not come.

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6. The flash does not take longer than three fifths of a second; the time is reckoned from the beginning of the flash.

On November 14, he reported that when he turned on the light at home he expected that a shock would come.

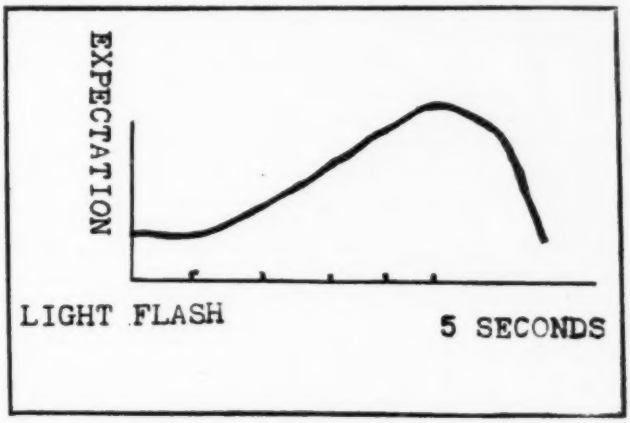
On November 16, he remarked that it was probable that he got his muscles ready when he expected the shock.

On November 17, in the twelfth experiment, no shock was given. He reported a feeling of disappointment and drew the accompanying curve, as the curve of his expectation.

On November 19, he drew a similar curve of expectation when the shock did not come. He spoke about a feeling of awareness. He remarked that he tried to forget the second stimulus.

On November 20, he touched the electrode by mistake before the flash was given and immediately had a feeling that the shock should come.

On November 21, he reported "I do not think of anything after the light has flashed, and I feel relaxed immediately after the light has come. Then I feel a slight increased tightening of the muscles in readiness for the second shock, but



Curve drawn by subject 1 to illustrate the curve of expectation he experienced.

no anxiety as in the first days of experimentation. Sometimes when I do not expect the shock in an attentive way, I feel it to be stronger than it is."

On November 23, he remarked that concentrated attention helped him to overcome the second stimulus and lessen its unpleasantness. He held his breath before the shock came.

Up to November 24 it had not been possible to get a conditioned reflex.

Summary.—1. The great expectation experienced during the first experiment disappeared very soon.

2. The greater tenseness in the first experiments led to many superficial associations. In the course of the experiments the superficial associations disappeared, and the subject concentrated on the shock; this concentration, he thought, made the shock seem less severe.

3. The concentration did not begin immediately with the flash of light, but developed immediately before the shock. A muscular preparedness was connected with that.

4. Conditioned reflexes were not observed.

5. The subject never had the impulse to withdraw the hand.

6. The shock was sometimes expected during the flash of light.

7. Similar objects (the electric light at home) provoked a similar expectation. Not only the light but also the electrodes conditioned the expectation.

Subject 2.—On October 26, in the first experiment, the subject, Dr. W., aged 36, believed that with the flash of light the experiment was over. The shock, therefore, made a stronger impression. In the second experiment he was tense after the flash. In the next experiment he was curious to see whether the experiment would change and whether the interval and the light were the same. He told about changes in the strength of the shock. In the eleventh experiment he had a dread of the shock and called himself a coward. In the fifteenth experiment he wondered how long the procedure would last. In the sixteenth experiment the shock seemed more painful by anticipation.

On October 27, he continued to wonder about the experiment. There was some dread before the shock. In the thirteenth experiment he experienced the desire to pull the arm away; he controlled it by will.

On October 29, in the sixth experiment, there was dread of the shock. He felt a tension before the shock came. In the sixteenth experiment he felt a tenseness before the shock, and a tightening of the muscles going as high as the elbow.

On October 30, in the first experiment, he wondered whether it would hurt. In the second experiment there was a suppressed desire to withdraw before the shock. During the fifth experiment he experienced some day dreams which he was not able to formulate. In the tenth experiment, he had a sensation in the hand before the shock came. Of the thirteenth experiment he said, "I didn't care for the shock."

On October 31, in the first experiment, there was a dread of the shock, with a tight feeling in the arm. In the third experiment there was a tightening in the hand. In the eighth experiment, there was tension before the shock. In the seventeenth experiment, the shock was delayed; there was no withdrawal.

On November 1, in the third experiment, there was a desire to withdraw. In the fifth experiment there were small movements of the fingers, but no withdrawal. In the sixth experiment he dreaded the shock and felt a tightening of the arm, "probably the desire to withdraw." In the sixteenth experiment there was the feeling of tension, with slight movements in the fingers (subjective and objective). In the nineteenth experiment, no shock was given. He did not withdraw the hand. He was astonished.

On November 2, there was anticipation of the shock. In the first experiment there was tension in the hand with a desire to withdraw it. In the second experiment he waited to see if the feeling came in the hand. In the fourth experiment, slight movements in the fingers were seen before the shock came. In the fourteenth experiment, the shock was delayed (six seconds). He looked at the hand, and felt a little movement. In the fifteenth experiment, he felt a tightening in the hand before the shock.

On November 3, in the fifteenth experiment, he wondered whether the fear was becoming less.

On November 5, he wondered whether he would experience a desire to take the hand away. In the fourth experiment there was dread of the shock. He remarked

that the tightening of the arm came when he expected the shock. Sometimes he had a feeling as if he could not stand it any longer. In the eighth experiment there was dread of the shock with tightening of the hand. In the ninth experiment there was tightening of the forearm also. In the twentieth experiment the shock was delayed; there was no withdrawal before the shock. From the subject's statements and from objective observation, it seems likely that in the moment when the shock is expected the hand is pressed with greater force against the electrode in order to prevent withdrawal.

On November 6, the subject experienced fear during the first experiment. In the seventeeth experiment, no shock was given. There was no conditioned reflex.

On November 7, in the fourteenth experiment, the subject tried to block off the feeling in the hand. In the sixteenth experiment, the fingers moved a little before the shock came.

On November 8, there were the same feelings of tightening and tenseness in the arm before the shock. A desire to withdraw the arm was present. Before the shock, this feeling sometimes became painful. In the fifth experiment the shock was delayed but no conditioned reflex was received.

On November 9, in the tenth experiment, there were slight movements in the fingers. In the seventeenth experiment, there was a strong desire to pull the arm away.

On November 10, in the sixth experiment, the shock was given later. No conditioned reflex occurred, but a tightening and slight movements took place at the time when the shock was expected (subjective and objective). In the nineteenth experiment, performed late (seven seconds), he reported that the tension lasted up to the time when the shock was expected and then diminished quickly.

On November 12, in the thirteenth experiment, the flash came a little later, after the hand had been fixed on the electrode. The subject had the feeling of expectation before the flash came, and the feeling of expectation disappeared with the flash, but then increased again.

On November 13, he reported that the feeling of tenseness did not come immediately after the flash of light but rather before the shock.

On November 14, in the seventh experiment, there was a little tremor in the hand. In the eighth experiment there was a desire to draw away the hand. In the eleventh experiment there was a dread of the shock. The subject raised the little finger before the shock came.

On November 15, in the third experiment, he felt a great deal of dread before the shock came, with a queer feeling in the hand, almost as if the hand would want to come off. In the fourth experiment he had a desire to jerk away with a dread of the shock as if the hand would be separated from the body. With experiment nine, there were slight movements in the fingers.

On November 16, in the fourth experiment, there was a desire to pull the hand away. In the ninth experiment there was a dread which seemed to be in the hand, combined with the desire to pull the hand away. In the twelfth experiment the subject felt a desire to pull the hand away. In the fifteenth experiment he tried to experience a feeling in his hand but could not.

On November 17, the shock was given two seconds later than expected, and the feeling of dread disappeared after five seconds. In the sixteenth experiment, immediately before the shock, there was a movement of the little finger. In the eighteenth experiment, no shock was given; he expressed a strong desire to pull away, a desire which disappeared after the fifth second.

On November 19, in the fourth experiment, the subject felt as if the hand would get away, and as if he could not hold it on. "The hand becomes personified,

as if it were alive, and as if it had its own feeling and movements." During this experiment slight movements in the fingers were evident, objectively and subjectively, before the shock came. In the seventh experiment, just before the shock came a definite, almost painful desire to get away. In the next experiment, the subject tried to change his feelings before the shock.

On November 20, in the thirteenth experiment, he experienced the desire to pull the hand away, with the feeling that his hand had contracted. In the seventeenth experiment he licked his lips immediately before the shock. In the next experiment he reported that immediately before the shock he felt a tension all over the body.

On November 21, the stimulus was delayed; a feeling of tension was strongly increased to the fifth second, and the shock came when he was almost relaxed.

On November 22, before the shock, twitching in the muscles occurred from time to time.

On November 23, in the fourth experiment, he began to think about his wife and was afraid that he would spoil the experiment by that. When he had other thoughts, the desire to jerk away the hand was not so strong. In the twentieth experiment, he complained about the strength of the shock. "One has to press down to prevent the hand coming up. It is like a depersonalization and connected with an almost painful sensation which increases to a crisis. It is as if the hand would jerk off." In the twenty-third experiment, sneezing distracted his attention during the interval. Neither the belated shock in the fifth experiment nor the complete absence of shock in the twenty-second experiment provoked conditioned reflexes. The subject always gave the same description. When the shock was expected, the tension immediately increased; it dropped when the shock did not come.

Summary.—1. The curiosity about the experiment disappeared as the experiment went on.

2. The feeling of dread, which was at first general, limited itself later to the end of the interval.

3. Although there was some general tension, the feeling of tension was felt especially in the arm, and was associated with muscular activities.

4. Objectively it was seen that before the shock the subject pressed his fingers down. The fibrillary twitchings observed very likely had something to do with a tendency to move the hand.

5. The tension disappeared soon if the shock did not come.

6. The tension became more and more localized in the arm. The arm was personified and in some way was separate from the personality. The subject spoke of depersonalization.

7. This depersonalization was in a close relation to counteracting tendencies, the one of getting away, which is not considered as belonging to the personality, and the other which tries to stay.

8. The subject compelled himself not only not to be a coward and to stay, but also not to think about other things during the experiment.

9. Once, this subject licked the lips immediately before the shock appeared.

10. The strong determination not to pull away the arm appeared as an outspoken movement and prevented the conditioned reflex.

SUBJECT 3.—On November 5, in the first experiment, the subject, Dr. K., aged 34, expected the shock and was curious about what might be going to happen. During the first experiment there came the free association, "Nacht und das Licht verschwindet" (Night, the light disappears). He had the feeling that the time interval had changed, although in reality there was no change. In the seventeenth experiment he remarked that he would be disappointed if the shock did not come.

On November 6, he felt the expectation and some sensation before the shock came, and in the third experiment of this day made the remark that he was better prepared. The motor reactions, which were strong on the first day and were reactions of the whole body, were less expressed. In the seventeenth experiment, he felt a tendency to withdraw his hand before the shock came. On November 7, in the seventh experiment, he felt impelled to take away the hand before the shock came. He felt an expectation and some kind of itching sensation in the hand. There was the same sensation in the thirteenth and fifteenth experiments.

On November 8, during the second experiment, he spoke of an increased expectation. In the eighth experiment suddenly the thought flashed through his mind, "Geduld allein" (patience only). During the eleventh experiment there came the thought, "Nicht das ich wüste" (I do not know), and during the twelfth experiment, "Ich weiss nicht was soll es bedeuten" (I do not know what it means). In the sixteenth experiment he thought, "Ist es noch nicht genug?" (Is it not enough?) In the same experiment, he felt a tendency to withdraw the arm before the shock should come.

On November 9, he came to the experiment with increased expectancy. The expectation diminished after the flashes of light and increased before the shock. During the seventh experiment there was again the thought, "Ich weiss nicht was soll es bedeuten" (I do not know what it means), and during the ninth, "Jetzt kommt's" (Now it comes). In experiment ten came the thought, "Licht, Liebe, Leben" (light, life, love). In the fifteenth experiment he thought, "Bald zu Ende?" (Will the end come soon?).

On November 10, he counted to himself during some of the experiments. In the seventeenth experiment, a little movement was seen in the hand before the shock came. He made remarks from time to time during the period of expectancy.

On November 12, at the second experiment, there came the tendency to withdraw the hand at the very moment when the light flashed. During the third experiment the word "warning" came into his mind when the light flashed; in another experiment he thought, "Where there is light there is much shadow," and in another experiment he quoted German poetry which is considered an example of complete nonsense. In the seventeenth experiment when, due to technical fault, the interval was eight seconds, he was not aware of it. In one of the experiments the German words came to him: "Vernunft wird Unsinn, Wohltat Plage" (Sense becomes nonsense, a good deed a nuisance).

On November 13, in the seventh experiment, there was the tendency to withdraw before the shock should come. It was difficult to keep the hand in position. In the experiments of this day expectation was increased, but he reported that he felt free in the first half of the interval and the expectation rose only toward the end of the interval. Only once did he feel a tendency to withdraw the hand at the moment when the light flashed.

On November 14, he complained about unrest in his arm toward the end of the interval. At the same time there was the tendency to withdraw. He spoke

about "local restlessness." During the flash of light in the twentieth experiment, he thought of the associations, "Light, lightning and Blitz."

On November 15, in the first experiment, he was preoccupied by thoughts he had had before. During the sixth experiment, he felt general restlessness. During the eleventh experiment there came with the light the word, "danger." During the fourteenth he had the tendency to withdraw before the shock should come and had to control this tendency.

On November 16, he felt, during the seventh experiment, a sensation before the shock came, but he felt the sensation more in the whole hand than on the skin. He felt an especial restlessness toward the end of the interval and pressed the fingers down strongly. During the tenth experiment there was a vague feeling in the arm. In the fourteenth, he repeated to himself, "Don't think anything." He remarked afterward that he had refrained from thinking any thought because he feared that any thought might interfere with the results. In the fifteenth experiment, the thought came "The moment of salvation will come soon."

On November 17, in the fifth experiment, he reported the tendency to withdraw his hand before the shock came. In the seventh experiment no shock was given. He withdrew his hand and asked, astonished, "Was there an electric shock?" During the following experiment he felt an increased expectation and an increased tension with which he combated the tendency to withdraw; in the twelfth experiment, when he did not receive a shock he kept his hand on the electrode and remarked, "I did not feel any shock but I did not relax."

On November 20, in the fifth experiment, there was a slight movement in the hand at the moment the light flashed. During the tenth experiment he felt a tendency to withdraw the hand, but during the thirteenth experiment, when no shock was given, he neither withdrew the hand nor felt the shock. During the seventeenth experiment, the thought came, "Bald zu Ende?"

In the following days, he remarked that he began to feel expectancy before the flashes of light, and once also that he expected the shock immediately in connection with the light.

On November 23, he remarked that the light made a double impression, one when coming and one when going. He said that sometimes it seemed as if the arm were not included in the general attention.

On November 24, he also expected the shock with the light. In the twentieth experiment no shock was given, but he did not withdraw and said that he did not expect the shock.

Summary.—1. In this subject it was noteworthy that there was at first a general curiosity and expectation which later disappeared. In this period, he believed that the interval was changed.

2. Before the coming of the shock vague sensations appeared in the arm, sometimes described as a restlessness in the arm. The general expectation of the first experiments changed into a localized feeling of restlessness in the arm. There was only one remark that his arm seemed not to be included in the general attention.

3. In the first experiment the expectation and tension were present during the whole interval. In the course of the experiments the first part of the interval became free from expectation, and the expectation began immediately before the shock.

4. After the first days of experimentation, the tendency to withdraw appeared. It was possible to get one conditioned reflex on the twelfth day of experimentation. After that day the tendency to refrain from withdrawal was increased so that in the next week no conditioned reflex could be obtained.

5. The shock was sometimes expected with the flash of light, once even before it.

6. In the period of expectancy, witty remarks expressed a criticism of the experiment which the subject did not feel in his conscious mind.

SUBJECT 4.—On November 5, just before the experiment began, the subject, Dr. C., aged 30, told of a dislike for electric shocks. During the first experiment he was much interested in all the little details of the room, the stop watch, etc. He began also to experiment with himself, whether he should fix his attention on the light or on the shock. He spoke sometimes in the expectation period about the dogs of Pavlov. He speculated whether the intensity of the shock or the time interval would change. His motor reactions were strong. He changed his whole attitude before he began with the experiment.

On November 6, in the first interval, he was apprehensive about the shock. During the ninth experiment, he withdrew the fingers when the light went on.

On November 7, he made an advance movement when the light flashed and moved his fingers before the real shock came without withdrawing them completely. During the fourth experiment he made a slight movement with the other hand when the light flashed, and during the eighth experiment he felt a desire to withdraw the arm. He had the feeling that the time interval changed, but in reality it did not.

On November 8, he made little movements with the other hand when the light flashed. In some of the other experiments he asked himself whether his pupils dilated. He had the idea that the time interval was about thirteen or fourteen seconds, and he counted to himself until the shock came.

On November 9, there was a movement of the right arm when the light flashed, but he did not completely withdraw the hand.

On November 12, he tried to withdraw the hand in the twelfth experiment before the shock came, but did not do it completely. In the seventeenth experiment, the shock came after six seconds. He did not remark about the difference and did not withdraw, but reported the impulse to withdraw the hand.

On November 13, during the first experiment, little movements of the little finger occurred. During the second experiment he made some movements with his finger which still remained on the electrode. In the fifth experiment he pressed his finger strongly against the plate before the light came. Also, in most of the other experiments there were little movements of the fingers, mostly in the sense of a strong pressure against the electrode immediately before the shock.

On November 14, during the second experiment, he concentrated on what was going on. He counted now "absolutely regularly." He was greatly interested in every movement of the observer, thought that it would be nicer if the observer would get a shock and felt that he should remain with his hands on the plate.

On November 16, he made little movements with the left thumb before the shock came. In the fifteenth experiment he remarked that the hand was tense and the muscles stiff. In the eighteenth experiment he made a movement of the hand before the shock came, but did not withdraw. He was greatly interested in what was going on.

On November 17, he continued counting. During the eleventh experiment, the hand sweated but seemingly he had had the perspiration before this experiment and was only more conscious of it during the period of expectation. Mostly, with the flash of light there were some changes in the whole attitude, especially in the fingers of the other hand. In the thirteenth experiment he did not get any shock, and yet he still lifted the hand. He said that he did this when he reached the number 14. "It was automatic. I was not thinking of the shock." In the fifteenth experiment he lifted the first two fingers from the electrode before the shock. He had counted to the number 13 and, as he said, "13 represents bad luck."

On November 19, he reported general tenseness.

On November 20, he did not get any shock in the twenty-second experiment. He did not withdraw the hand after five seconds but withdrew it about two seconds later "in order not to get the shock later."

On November 22, at the fifteenth experiment, he withdrew his hand completely without any shock. He was counting. "If I would not count I would not draw away. I felt that the shock ought to be here." He felt rather silly afterward because he withdrew his hand. In the next experiment he counted, as he said, verbally, and licked his lip immediately before the shock came. In the twenty-first experiment, he moved his fingers a little before the shock came.

On November 23, he remarked during the first experiment, "I dread so much the first shock that it is a real effort to keep the hand against the electrode, against the involuntary tendency to take the hand away."

In the fourth experiment, the shock was delayed. He had the tendency to withdraw the hand. He still continued counting in order to eliminate the suspense. "I like to be under conscious control." During all the experiments, he pressed his hands down as overcompensation. In the eighteenth experiment, he first lifted and then pressed down his hand. In another experiment made on the same day, he developed the idea that one might control the pupils by electric shocks.

In the fourth experiment on November 24, he felt the tendency to move away and to lift the arm before the shock should come, and, indeed, lifted the fingers from the electrodes but in such a manner that he still received the shock. In the twenty-sixth experiment no shock was given. He did not withdraw the hand and said that he had reckoned that on this day the shock would not come.

Summary.—1. This subject took a special motor attitude before the beginning of every experiment.

2. With the flash of light often came motor reactions, at first in the right hand and later in the left.

3. In the first period of experimentation the greater curiosity was shown again in ideas about changing the interval.

4. In the course of the experiments, the subject began to count in order to know when the shock would come.

5. Before the shock came, there often occurred involuntary movements which were counteracted by the opposite effort. The subject reported a tendency to withdraw the hand. Later, little movements occurred.

6. The conditioned reflex was clearly manifested twice. The subject tried to excuse himself and made use of a pretext. Once, instead of a real withdrawal, a partial withdrawal took place.

7. Once the subject withdrew the hand later, in order not to get the shock.

8. The subject excused himself for withdrawing.

9. Counteraction against the tendency to withdraw was obvious.

10. Once, the subject licked his lips when the shock came.

SUBJECT 5.—On October 25, in the first experiment, Miss T., aged 32, experienced curiosity about the details of the experiments. In the first experiment, the current was too weak and had to be reinforced. The subject wondered whether the light would be different. She was bored by the repetition (October 25).

On October 26, the subject sometimes forgot that the shock would come. She was bored by the repetition in the experiment.

On October 27, in the sixth experiment, there was a general feeling of anxiety.

On October 29, she remarked that she also felt an increased susceptibility to noises during the day. In the tenth experiment she felt an inclination to pull the arm away. In the fourteenth experiment she had difficulty in keeping the arm on the plate—a tendency to withdraw. She was disappointed because there was no variation in the experiment.

On October 30, in the twelfth experiment, the sensation was felt before the shock came. In the fourteenth experiment she concentrated on the light and did not feel the sensation in the hand. In the next experiment, she tried to suppress the feeling but could not. In the next experiment, she concentrated again on the light in order to feel less.

On October 31, in the eighth experiment, she said "I have to work against the impulse to withdraw." She complained when the experimenter hesitated a little with the flash of light. She already felt worried when she put her hand on the electrode. In the thirteenth experiment she complained "how disagreeable the whole situation is." In the fifteenth experiment she moved before the shock came and said, "I cannot keep the hand there any longer. I cannot help it." In the eighteenth experiment she was impatient before the shock. In the nineteenth, there was a feeling of numbness in the fingers. She complained that control was impossible.

On November 1, there was a greater effort to concentrate, and no withdrawal occurred. In the seventeenth experiment, during the interval, the word "numbness" came into the mind, but no numbness was felt.

On November 2, in the fifth experiment, she said "I do not want to keep the hand there longer." She complained about difficulties in keeping the hand on the plates. In the next experiment no shock was given. She withdrew the hand with the remark "I could not keep it there." In the following experiment, the subject pressed strongly against the metal (subjectively and objectively) in order not to withdraw. In the ninth experiment the hand trembled before the shock. In the tenth experiment she said: "It is not psychic, it is a motor reaction." In the eleventh experiment there were general tenseness and sensitiveness before the shock came. In the fourteenth experiment there were difficulties in keeping the hand on the plate, and she moved the fingers a little. In the next experiment, the expectation was increased. In the seventeenth experiment no shock was given; the hand was withdrawn.

On November 3, she complained that the tenseness began not at the moment of the flashes of light but at the moment when the electrode was touched. In the first experiment, a real effort was made to keep the hand there. In the third experiment there was a sensation of numbness in the hand while waiting. In the fifth experiment there was a conflict between the tendency to withdraw and to keep the hand there. In the next experiment there was increased trembling before the shock; "I want to pull the hand away." In the eleventh experiment, no shock was given. She had a disagreeable sensation in the hand and withdrew it. In the twentieth experiment the subject had the impression that she lifted the hand before the shock came; "It seems I can withdraw just before the shock comes."

On November 5, in the first experiment, the feeling was present that "it lasts a long time." In the fourth experiment the feeling of numbness seemed to diminish the intensity of the shock. In the tenth experiment, no shock was given; the hand was withdrawn. The feeling of numbness came: "I could not press it down. I resent it when I withdraw." In the next experiment there was the impulse to withdraw before the shock came but it was not followed out. In the next, there was a little movement but not complete withdrawal before the shock. In the next experiment she had the determination not to move. In the fourteenth experiment, trembling took place before the shock came.

On November 6, there was strong anticipation. In the sixth experiment the fingers were withdrawn after three seconds. In the thirteenth experiment there was a sensation "as if the hand would lift itself." In the fifteenth experiment a little movement occurred before the shock came. In the seventeenth experiment there was conflict and definite effort to keep the hand in place.

On November 7, in the second experiment, there was numbness before the shock came. In the sixth experiment she had great difficulty in keeping the hand in place. In the fourteenth experiment, the shock was given after six seconds; there was no withdrawal. In the eighteenth experiment, no shock was given; the hand was withdrawn; there had been a numb sensation in the hand preceding the withdrawal which was "just uncontrollable."

On November 8, in the fifth experiment, there was increased numbness. In the seventeenth experiment, no shock was given; there was tension with increased numbness; the hand was withdrawn in the fifth second.

On November 9, movement of the hands occurred before the shock came, but there was no withdrawal. In the fifth experiment she had the feeling that as a result of the numbness the contact with the electrodes was lost. In the seventh experiment she moved a little before the shock came. In the ninth experiment the shock was delayed; there was no withdrawal. The same was true in the sixteenth experiment. In the twenty-first experiment a shock was not given; in the fifth second she withdrew the hand, saying "I could not hold it, it was so intense, the feeling of numbness. I wanted terribly to pull my hand away. It does not begin immediately after the light flash; it rises gradually, reaches a peak and then is withdrawn."

In retrospect, the subject repeated that her tenseness began at the moment when she touched the electrodes.

Summary.—1. In the beginning, there was interest in the experiment and in the question of variation in it. The subject felt bored because the experiment did not change.

2. The pain gave a general discomfort which came out in the increased sensibility to noises.

3. Soon the tenseness concentrated on the experiment. The anticipation provoked a feeling of numbness in the fingers.

4. This numbness was felt as a protection against the shock.

5. Soon a strong tendency to withdraw appeared, which led in the following days to a conditioned reflex.

6. Sometimes one found a premature reaction.

7. Not only the flash of light but also the electrodes had a conditioning result.

8. Instead of the feeling of numbness, once there came the word "numbness."

9. The conditioned reflex made the subject ashamed, and she increased her efforts to keep the hand down.

10. During the experiment, the subject sometimes fixed her attention on the light, sometimes on the shock.

11. The hand was in some way independent "as if it would lift itself."

COMMENT

1. In all my subjects there was at first a great expectation and curiosity about the experiment, which disappeared in the course of the experiments. When conditioned reflexes occurred, they appeared only after the first curiosity was gone. In this period of curiosity the subjects often believed that the experimenter varied the experiment, but in reality this did not occur. It is true that the induction apparatus varies a little in intensity, and some of the subjects begin to sweat in the palm of the hand during the experiments, but the other factors were constant. Nevertheless, the subjects believed that some variation in the interval and in the light took place.

2. In the course of the experiment, the attention of the subject was more and more concentrated on the experiment as such. In two of the subjects, many thoughts occurred during the first days of the experiments. In the case of subject 1, one received the impression that these passing ideas disappeared when the shock was less dreaded. In the case of subject 4, some labile attention for the objects in the room took place. Very likely, in both subjects one dealt with superficial observations and thoughts which helped to overcome the disagreeable expectation, a mechanism which might be on the basis of some manic reactions with flight of ideas, and which can be observed during psychoanalysis when a person who has made a transference does not want to speak about it and begins with a superficial interest in the little things in the room.

3. In one subject only (subject 4), before the beginning of every experiment some motor adaptations became visible. Subject 2, however, spoke also about the general tension. Very likely the other subjects would have reported something similar if they had been asked.

4. In all subjects, in the beginning there was a great fear of the shock. Subjects 1 and 2, especially, who did not show conditioned reflexes, gave a clear report that in the course of the experiment the expectation and fear came only immediately before the shock and that the flash of light as such did not cause the feeling of dread. This feeling reached its height at the time when the shock was expected and disappeared when the shock was not given at the expected time, a fact which is the more remarkable because in most of the experiments the shock was given later. Whereas in one of those subjects the expectation was a more general feeling, in the other subject there was in some way a more localized feeling. This subject had a strong motor tendency to withdraw, which was associated with little movements which appeared also in the experiment. It is interesting that in connection with those muscle activities a localization of the feeling takes place, and this is a fact which points to the general theory that feelings are in connction with special activities of the body and are not so different from sensations as one usually supposes. In this case the feeling, of course, is not a cause of the special tendencies and can be interpreted in a clear way as the consequence of tendencies which also find their muscular expression. Subject 3 also spoke about localized restlessness in the arm.

5. Subject 2 felt that the arm did not belong to his own personality while this conflict between two tendencies went on. That is indeed an experimental approach to the problem of depersonalization, a state of mind which, according to my previous statements, is in connection with the conflict between counteracting tendencies. In the observation, reported, depersonalization was partly the expression of a self defense against the shock. The shock shall not belong to the personality. But it was also a self defense against tendencies of which the subject did not approve. In the subjects in whom the conditioned reflex took place there was a feeling of shame, and the subjects excused themselves and said that they had not moved the hand themselves, the hand had moved, it was automatic, and so on, tendencies similar to those which led to depersonalization in subject 2. Very likely the feeling of numbness in the case of subject 5 had a similar significance, and the subject also made the remark that it was as if she would feel less because of the numbness.

6. Most of the subjects often change the direction of their attention. It is partly a psychic play they perform, but, as shown by the remarks of subject 2, it is partly also a mechanism of self defense and belongs in the same order as the scattering of attention away from the painful sensation.

7. In two of the subjects no conditioned reflexes were obtained, if one considers as a conditioned reflex the motor reaction of withdrawing the hand from the electrodes. In both these subjects, however, typical psychic processes have been conditioned, which in subject 2 found a clear

motor expression. In the case of subject 1 only expectancy was created, and the impulse to withdraw did not occur. In subject 2 the conditioned desire to withdraw was very strong.

8. In the three subjects in whom conditioned reflexes were observed in a typical way, the coming of the typical reflex was always preceded by a tendency to withdraw; in subject 4, who also spoke about a desire to withdraw, there were sometimes some imperfect movements in the direction of withdrawing. Sometimes, movements of the little finger were rather obvious. In the case of subject 3, the conditioned reflex was preceded by sensations in the arm and also by restlessness in the arm. The localization of the feeling also is quite similar to that in the case of subject 2. In subject 5, the relation of the conditioned withdrawing to the tendency of withdrawing was obvious, and the tendency to withdraw sometimes led to too early reaction. All the subjects excused themselves by saying that the withdrawing was involuntary. Subject 3 was astonished and asked whether there had been any shock. After the withdrawing, the subjects felt ashamed and increased the tendency not to withdraw. Also, the withdrawing was always felt as an action of the personality, but as an action which the subject is directed against. There is a complicated struggle between two tendencies in the three persons who showed conditioned reflexes as well as in those who did not come to a real motor response. The principal psychic mechanisms in all subjects are identical. There is one primitive reaction which, in connection with self preservation, makes the person withdraw in a moment of danger, and there is another in connection with the so-called moral part of the personality in relation to the task to which the person puts himself which does not allow the primitive tendencies to come out. The subject feels some feeling of guilt when the primitive tendency breaks through. I do not believe that it is possible to describe what is going on in the experiment in a simpler way. I can only say that there is a situation of a special kind, that the subject develops a special attitude toward a situation which repeats itself always in the same way, and sets himself tasks also when a special task is not asked for. I do not believe that experiments with dogs have any other basis, but it is not possible to get en rapport with animals. On other occasions I have developed the general idea that one will not understand human behavior better by studying the objective reactions of animals; but one will understand the psychology of the objective reactions of animals better after having studied carefully the psychic attitudes of human beings in the different levels.

9. The result of the conflict between withdrawing and keeping the hand down is sometimes an increased keeping down, as, for instance in the case of subject 2, and in a similar way also in the cases of subjects

4, 3, and 5; or a partial withdrawal, withdrawing some fingers, mechanisms which are well known as mechanisms in the conflict of two opposing psychic tendencies.

10. In two of the persons who have shown marked conditioned reflexes and in subject 2 some reproduction of the electric stimulus was felt, but this reproduction was incomplete. In subject 3, it led to the astonished question, "Was there any shock?"

11. The withdrawing in the conditioned reflex is certainly in a close relation to the entire motor adaptation of the person. This motor adaptation came out in an especially clear way in subject 4, but it is very likely present even in the negative experiments with subject 1, who complained about disappointment when the shock did not come. One can also observe that the subjects in some way become more and more skilful in their movements of withdrawal. The movements become more deliberate and less exuberant. The motor adaptation leads to an increased readiness to withdraw. With this readiness, helped by fear and expectation, the desire to pull away breaks through and the conditioned reflex appears.

12. In all of my subjects, the conditioning went as far as the purpose of the experiment. In the negative case, subject 1, a similar impression, the turning on of a light at home, provoked the expectation of the shock. For many of the subjects the touch of the electrodes conditioned whether the light flashed or not. This was clearest in subject 5, but it was also to be observed in subjects 3 and 1. Under the influence of psychic tendencies one knows that one cannot cut the situation into its component parts, but one reacts to it in a more diffuse way.

13. The psychic tension of the subjects creates, of course, a special attitude of mind. In this attitude of mind the time relations are not evaluated as they would be without the tension. Subject 4, at first, often reacted with movements of the right hand immediately with the flash of light. After the experimenter remarked about this movement of the right hand, this movement was transferred to the left hand. Almost all the subjects from time to time had the feeling that the shock should come with the flash of light. One can say generally that the expectation leads to displacements in time, a mechanism which one knows in the so-called unconscious thinking. It is remarkable that subject 3 also showed other signs of so-called unconscious activity. Against his will, German sentences which illustrated a criticism of the experiment came into his mind. The sentences were well chosen and witty. That is a hint that during this period some special state of mind was present. Subject 5 once suddenly thought of the word "numbness" without the feeling of numbness.

CONCLUSIONS

The experiments make it clear that one is dealing with complicated psychic processes and adaptations. The phenomena studied here are the same phenomena one observes in animals. I want only to hint at what, for instance, Beritoff wrote about generalization and differentiation of the conditioned reflexes. One would have to see generalization of the conditioned reflexes in the fact that the turning on of the light provoked the expectation of the shock and that also the touch of the electrode was conditioning. Very likely this generalization would have disappeared if the experiments had been continued. Beritoff also mentioned the fact that the reflex begins to appear sometimes in the way of a slight movement of the leg. I have observed similar results in my subjects. I do not want to go into details. I want only to emphasize the fact that there is no principal difference between the conditioned reflex of animals and the conditioned reflex of man—a point of view which is also held by Lurja.[7] It can be supposed also that during the conditioning of reflexes in animals similar psychic processes go on.

7. Lurja: Die moderne russische Physiologie und die Psychoanalyse, Ztschr. f. Psychoanalyse, 1926, vol. 12.

PROLIFERATION OF THE ARACHNOID CELL IN AND AROUND THE DURA MATER*

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I wish to present here certain observations that were made in a gross and microscopic study of the dura mater removed at necropsy in fifty-nine cases in the Mayo Clinic, which, as correlated with certain clinical observations, are suggestive as to the source of endotheliomas.

The gross examination of the dura mater, stripped from the skull and handled with care, showed, in a large percentage of cases, small nodular or vesicular outgrowths, which were found often in juxtaposition to the meningeal vessels, as well as in other parts of the dural surface (figs. 1 and 2). This is not a new observation. Observers[1] in the middle of the last century called attention to similar peculiar cellular proliferations, the etiology and complete significance of which still retain important elements of conjecture despite more recent excellent contributions to the subject.[2]

Microscopically, it was found (fig. 2) that these nodular or vesicular structures possessed, in general, dural fibrous tissues, a certain amount of areolar tissue, connective tissue, and, as a rule, characteristic groups of cells which were identified as the type of cell (figs. 3 and 4) found

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1. Cushing, H.: The Cranial Hyperostoses Produced by Meningeal Endotheliomas, Arch. Neurol. & Psychiat. **8**:139 (Aug.) 1922. Meyer, Ludwig: Die Epithelsgranulationen der Arachnoidea, Virchows Arch. f. path. Anat. **17**:209, 1859.

2. Cushing, H., and Weed, L. H.: Studies on the Cerebrospinal Fluid and Its Pathway: IX. Calcareous and Osseous Deposits in the Arachnoidea, Bull. Johns Hopkins Hosp. **26**:367, 1915. Essick, C. R.: Formation of Macrophages by the Cells Lining the Subarachnoid Cavity in Response to the Stimulus of Particulate Matter, Contrib. 272 Carnegie Inst., Washington **9**:377, 1920. Schmidt, M. B.: Ueber die Pacchionischen Granulationen und ihr Verhältniss zu den Sarcomen und Psammomen der Dura Mater, Virchows Arch. f. path. Anat. **170**:429, 1902. Weed, L. H.: The Pathways of Escape from the Subarachnoid Spaces with Particular Reference to the Arachnoid Villi, J. M. Research **31**:51, 1914-1915; An Anatomical Consideration of the Cerebrospinal Fluid, Anat. Rec. **12**:461, 1917; The Cells of the Arachnoid, Bull. Johns Hopkins Hosp. **31**:343, 1920. Winkelman, N. W., and Wilson, George: Localized Proliferations of the Arachnoid; Possible Relation to the Origin of Tumors, Arch. Neurol. & Psychiat. **14**:46 (July) 1925.

Fig. 1.—Cranial surface of dura mater. A vesicle-like growth of arachnoid cells is indicated by the arrow.

Fig. 2.—Vesicle-like growth of arachnoid cells which has broken through the dura mater; × 18.

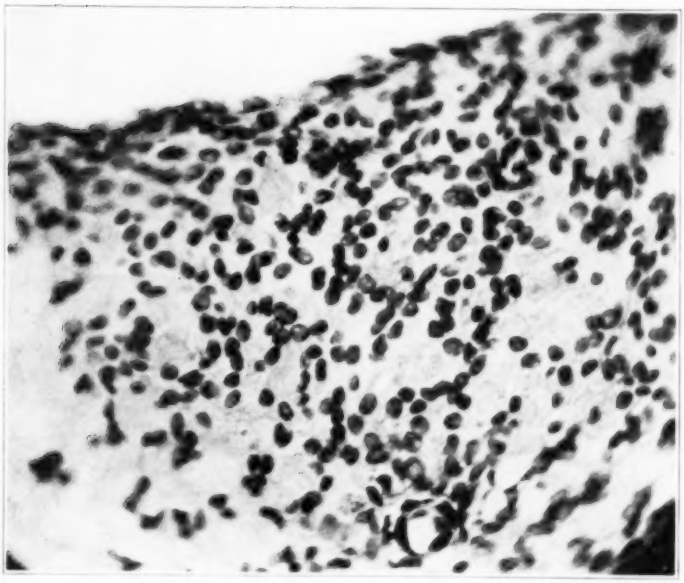

Fig. 3.—Grouped arachnoid cells; × 350.

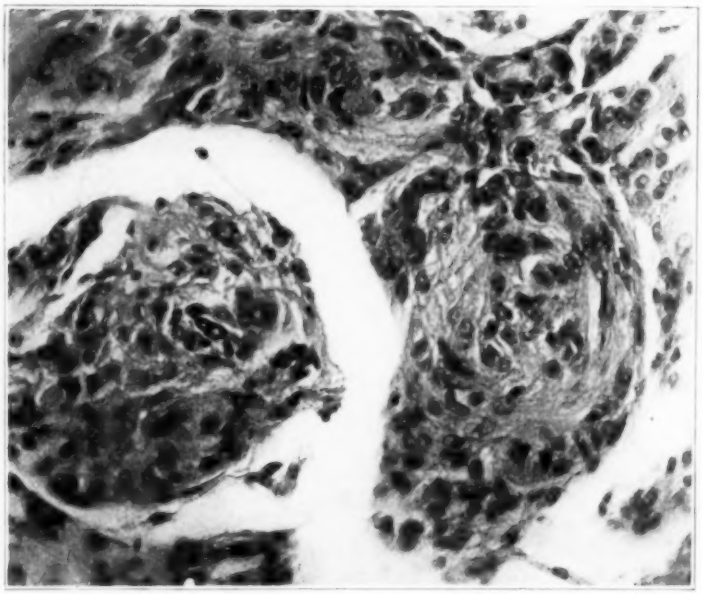

Fig. 4.—Cells from a tumor diagnosed as endothelioma. Arachnoid cells are shown; × 350.

in endothelioma.[3] These cells have large, round or oval nuclei, stain deeply with hematoxylin, and have scant protoplasm. As seen on the surface of the arachnoid, they are low, cuboidal, endothelial-like cells. The cell outline is not demarcated by the ordinary stains. The typical arachnoid cell is generally elongated. It is pale and translucent, with a faint nucleus.[4]

The origin of such cell growth within the dura mater has been a moot question, as the so-called dural endotheliomas are supposed to arise from a cell layer lining the inner or cerebral aspect of the dura, presumably endothelial cells.[5] But the cells comprised in these small

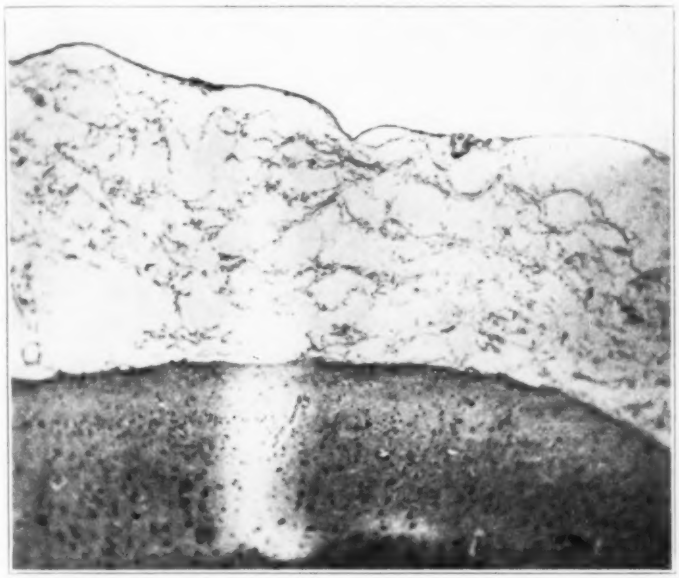

Fig. 5.—Arachnoid and adjacent cerebral cortex. Areas of slight thickening on the arachnoid surface are shown; × 18.

growths are not endothelial cells, and furthermore there is no cell lining on the inner aspect of the dura mater.[6] The only so-called cell lining which may come in contact with the cerebral surface of the dura mater is provided by the arachnoid; and since the arachnoid membrane on its

3. Mallory, F. B.: The Type Cell of the So-Called Dural Endothelioma, J. M. Research **41**:349, 1919-1920.

4. Kubie, L. S., and Schultz, G. M.: Vital and Supravital Studies of the Cells of the Cerebrospinal Fluid and of the Meninges in Cats, Bull. Johns Hopkins Hosp. **37**:91, 1925.

5. Mönckeberg, J. G.: Endotheliom, Ergebn. d. allg. Path. u. path. Anat. **10**:789, 1906.

6. Weed (footnote 2, fourth and fifth references). Mallory (footnote 3).

outer surface is entirely separated from the inner surface of the dura mater,[3] the difficulty of solving the problem apparently increases.

The arachnoid is derived from the mesenchyme surrounding the brain and spinal cord. It differentiates early in embryonic life, appearing first as a single layer of cells, which later increases to several layers in thickness, ranging from two to five cells in embryos of from four to five months. These cells at first lie close to the inner or cerebral layer of the dura mater, but later separate from it, leaving the dura without a lining membrane. After this separation, the outer cells of the arachnoid, facing the dura mater, form a layer of endothelial-like cells, which are really undifferentiated but potential fibroblasts and form the surface lining of the arachnoid.[3] The arachnoid thus is comprised of a single layer of cells facing the cerebral aspect of the dura mater and in contact with it at various places; and from the reticulum, which pro-

Fig. 6.—Arachnoid cells breaking through the dura mater; × 9.

vides its framework, numerous trabeculae covered by arachnoid cells extend to the pia mater (fig. 5).

That arachnoid cells themselves multiply irregularly with age, forming local proliferations here and there with areas in which there is only one layer of cells forming the membrane (fig. 5) has been shown,[7] and the conclusion seems to be that this is a normal growth, although the possibility of inflammatory and traumatic influences in stimulating such proliferations has been suggested. When arachnoid cells are found not only in contact with the dura mater but also clustered in groups against it, and at times growing into (fig. 6) and even through the dura mater (fig. 2), the possibility of abnormality seems more certain. This type of growth has been attributed to dural defects through which the arachnoid cells are forced by a long-continued moderate

7. Essick (footnote 2, second reference). Winkelman and Wilson (footnote 2, seventh reference).

increase of intracranial pressure, to chronic inflammation, to trauma and to the presence of various diseases. The common etiologic factors of neoplasms in general are thus offered as explanatory, as well as a number of other obscurely related conditions.

Increasing age has been mentioned so often [8] as an etiologic factor that it has become almost an assumption that proliferations of the arachnoid do not occur in the young. The present series of cases does not substantiate this statement. It may be well to call attention to the finding of whorl formations (figs. 7 and 8) in half of the cases of the first age group, as recorded in the accompanying table.

The relationship of increased intracranial pressure to the presence of arachnoid proliferations seems fairly definite. Growths were present

A Summary of the Clinical and Pathologic Data on the Arachnoid Proliferation in Fifty-Nine Cases

	Total Number of Cases		Number of Cases with Increased Intracranial Pressure		Arachnoid Cell Clusters						Benign Whorls			
					With Intracranial Lesion		Without Intracranial Lesion		With Increased Intracranial Pressure		With Increased Intracranial Pressure		Without Increased Intracranial Pressure	
Age, Years	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
0–20	9	5	8	3	6	4	..	1	6	3	4	..	..	1
21–40	8	..	4	..	5	..	1	..	4	..	2	..	..	..
41–60	17	11	7	5	11	5	3	1	6	2	1	..	2	1
61–80	8	1	1	..	3	1	2	..	1	..	..	..	..	..
Total	42	17	20	8	25	10	6	2	17	5	7	..	2	2

in twenty-two of the twenty-eight cases in this category. In eighteen of the cases, the intracranial tension was due to tumor of the brain; in the remaining ten, to meningitis, hydrocephalus, cerebral hemorrhage and abscess of the brain. When intracranial tension is increased, the arachnoid is undoubtedly forced into closer contact with the dura mater. There is a further possibility that the cerebrospinal fluid under the increased tension may favor hypertrophy of the arachnoid membrane, and especially of the villi, since it had been shown [9] that the villi function in the escape of the fluid. Whether the villi are active or passive in this function is open to question.

Insanity, long-standing epilepsy, general paralysis and senility were among the first mentioned etiologic factors in the production of "arachnoid granulations." [10] There were ten cases of the series that might

8. Weed (footnote 2, sixth reference). Winkelman (footnote 2, seventh reference). Meyer, Ludwig: Ueber die Bedeutung der Pacchionischen Granulationen, Virchows Arch. f. path. Anat. **19**:171, 1860.

9. Weed (footnote 2, fourth reference).

10. Meyer (footnote 1, second reference).

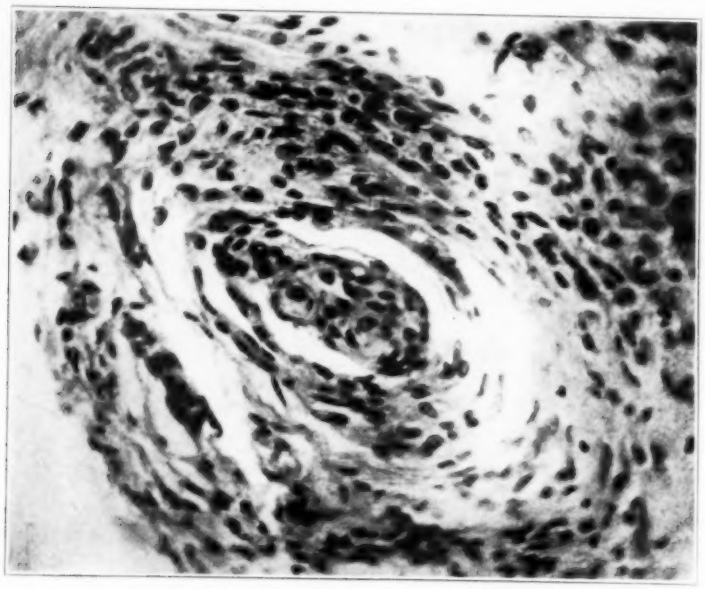

Fig. 7.—Whorl formation; × 350.

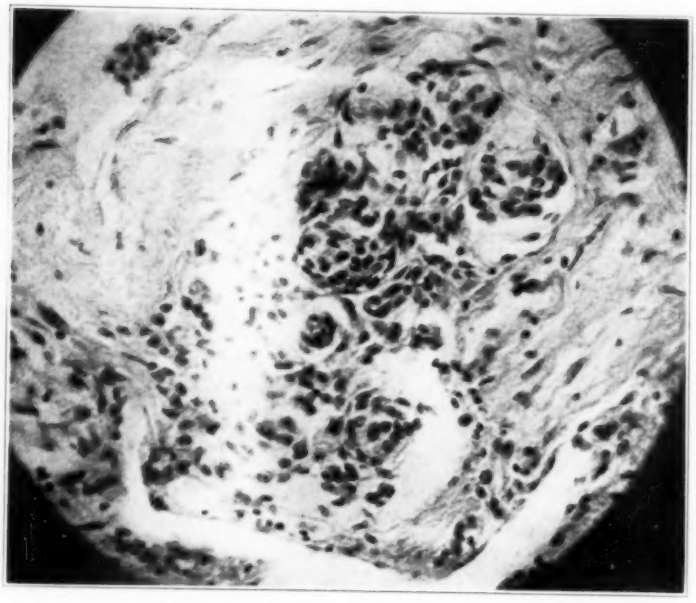

Fig. 8.—Whorl formation; × 300.

be thrown into the heterogeneous grouping of "insanity" and "psychosis," and in eight of these the arachnoid growths were adherent to the dura mater.

Chronic inflammation (including infection) has been mentioned as an etiologic factor.[11] In only three of the cases was the meningitis definite; one case of abscess of the brain may possibly fall in this group, since all cases showed arachnoid proliferations.

The suspected relationship of multiple fibromatosis involving the central and peripheral nerves to endotheliomas [3] is founded on the fact that both types of growths have occurred at one and the same time. As one of the cases of the present series was diagnosed "neurofibromatosis," a careful examination was made of the dura mater, but all that was found was a yellowish plaque which, on microscopic examination, proved to be fibrous tissue only.

In eleven cases, no clinical or anatomic cerebral or intracranial lesions which grossly could be construed as influencing the production of local arachnoid proliferations were present. Even such cases as pernicious anemia, aplastic anemia and pellagra are excluded from this group. In only three of this group were clusters of arachnoid cells observed macroscopically (fig. 1). In five other cases there was a decided thickening of the dura mater (fig. 6) in certain areas, which, on microscopic examination, revealed an arachnoid cell growth. Thus, in eight of the eleven cases, dural growths in which arachnoid cells were more or less involved were present, which is sufficient evidence that such growths may be found in the normal person.

Several points of similarity of arachnoid cell proliferations in the dura mater to the "dural endotheliomas" can be confirmed. As mentioned, the two growths have been shown to possess the same type of cell.[3] In the present series mitosis was not found, and as a consequence it may be concluded that active hyperplasia was not present.[12] Whorls (figs. 7 and 8) were found in eleven of the fifty-nine cases. These whorl formations were similar to those found in endotheliomas (fig. 4), and the whorls were usually single, but rarely there were two or three in a group. Furthermore, the whorls did not characterize all the groups of arachnoid cells, which usually consisted of many cells without orderly arrangement. Occasionally, a suggestion of palisading of nuclei was noted. A certain number of the arachnoid growths passed through the dura mater (fig. 2). These came in direct contact with the skull and could readily be assumed to mark the beginnings of that group of endotheliomas which grow up through the skull and form the areas of

11. Meyer (footnote 1, second reference). Winkelman (footnote 2, seventh reference). Cleland, J.: Description of Two Tumors Adherent to the Deep Surface of the Dura Mater, Glasgow M. J. **11**:148, 1863-1864.

12. Weed (footnote 2, sixth reference).

hyperostosis occasionally seen in the presence of such growths.[13] Endotheliomas with hyperostosis comprise about 20 per cent of all dural endotheliomas,[13] although certain authors believe this association is rare.[14] The number of arachnoid growths extending outward toward the skull could not be approximately computed. Calcification was found in only two or three instances in the series and may be considered negligible. It has been stated that the older the animal the more calcified nodules are to be expected,[15] but I could not confirm this.

In regard to the function of clusters of arachnoid cells, this study has nothing to offer except to appreciate, in passing, that Weed's experiments [9] indicate that they are passageways for cerebrospinal fluid. It may be noted also that similar clusters of cells with similar functions have been found in the spinal subarachnoid angle.[16]

CONCLUSIONS

1. Proliferation of arachnoid cells facing the dura mater are found at every age and in normal as well as abnormal intracranial conditions.

2. These proliferating cells may merely be in contact with the dura mater, or may pass into or through it, in certain places coming in contact with the adjacent skull.

3. Groups of these proliferating cells often have characteristic whorls such as those found in endotheliomas.

4. Arachnoid outgrowths are often but not always found associated with increase in intracranial pressure.

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13. Cushing (footnote 1, first reference). Penfield, W. G.: Cranial and Intracranial Endotheliomata; Hemicraniosis, Surg. Gynec. Obst. **36**:657, 1923.

14. Krause, Fedor: Surgery of the Brain and Spinal Cord, Based on Personal Experience, New York, Rebman Company, 1912.

15. Cushing and Weed (footnote 2, first reference). Essick (footnote 2, second reference). Weed (footnote 2, sixth reference).

16. Elman, Robert: Spinal Arachnoid Granulations with Especial Reference to the Cerebrospinal Fluid, Bull. Johns Hopkins Hosp. **34**:99, 1923.

PSYCHOGALVANIC STUDIES OF NORMAL AND OF ABNORMAL SUBJECTS

FOR THE PURPOSE OF DETERMINING THE RELIABILITY OF THE GALVANOMETER IN INVESTIGATING THE EMOTIONS *

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THE PSYCHOGALVANIC REFLEX

The galvanometer as an instrument for the study of mental and emotional states has been in use for some time, but never has it aroused the interest that it does today. It is difficult to discover the precise reason why such unusual attention has been given to this type of psychologic experimentation, unless it is a feeling that here at last is a method of studying emotional or affective states in a quantitative manner. Psychology has always recognized that to understand conduct in any thorough manner it is necessary to know more about the urges and drives, or the feeling side of human nature. The psychogalvanic reflex seems to be definitely related to emotional states. If this can be demonstrated and the differences in emotional reactions can be quantitatively measured, remarkable strides forward in the study of behavior will be possible.

Work with the galvanometer, begun in 1924, at the Pennsylvania Hospital, was undertaken to determine whether the galvanometer offers a new and improved method for the study of the emotions. This paper is the first report on the results and the technic used. After the apparatus began to function satisfactorily, the next step was to verify, if possible, the results and claims of other experimenters. This phase of the work required much time and changing in order to test some of the methods employed by other workers, training of an assistant to take records and, finally, the devising of a satisfactory technic.

It should be added that as the experiment progressed it became evident that the set-up could be used for an experimental study of emotional states in the mentally diseased. A second article covering this phase of the work is in preparation.

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APPARATUS [1]

After many months of experimentation, the following apparatus was adopted:

1. Mirror torsion galvanometer, Leeds and Northrup, catalog no. 2239 A. 106071, sensitivity 85 megohms, upper suspension 0.003, phosphor bronze strip, resistance 119 ohms.

2. One ground glass scale, set up at a distance of 29 inches (73.66 cm.) from the galvanometer.

3. Electric light bulb and lens.

4. One Ayrton shunt (Inf.: 0; 0.0001; 0.001; 0.01; 0.1; 1).

5. One Leeds and Northrup enclosed dial Wheatstone bridge, catalog no. 4760 (four dial decades: 9 [1 10 100 1,000] ohms, ratio arms: single dial control; seven multipliers: 0.001, 0.01, 0.1, 1, 10, 100, 1,000.)

6. One "Everready Columbia" dry cell.

7. Nonpolarizable electrodes made by attaching zinc plates to a copper wire, which in turn are inserted into glass tubes containing shredded asbestos washed in acid and soaked in saturated zinc sulphate solution. These glass tubes are then dipped into glass vessels containing a saturated solution of zinc sulphate. The glass vessels contain a no. 3 porcelain Gooch crucible (perforated bottom) which is suspended by strings attached to holes in its sides. The outer glass receptacle is then placed in a larger glass jar filled with water of the proper temperature. (Into the porcelain crucible the subject dips the forefinger of each hand up to the first joint.)

8. Stop watch and record blank.

The galvanometer is attached to a board which is fastened to the wall of the room and can be adjusted vertically and horizontally. Wires run from the galvanometer to the shunt on a table which also supports the electric bulb holder and lens, the Wheatstone bridge, battery and scale. The experimenter sits at the end of the table so that he can balance the bridge and see the subject and the scale without changing his position. There is a screen at the left of the table which cuts all view of the apparatus from the subject. The subject sits in a comfortable arm chair with specially designed arm rests, near the end of which are holes opening into the vessels containing the zinc sulphate solution. Between and a little behind the subject's chair and that of the experimenter is another chair occupied by the assistant. A shaded electric light is fastened to the left side of the screen which illuminates the subject and the board for the record blank in the assistant's lap.

PROCEDURE

The temperature of the zinc sulphate solution and the room temperature are taken before each subject comes into the room. The temperature of the fingers is taken in an adjoining room, and on entering the experimental room the subject is seated in the special chair. The following instructions are given:

"We want you to help us make a little test to see how much better you are. There is nothing here to hurt you, and it will take but a short time. All you must do is to put the first finger of each hand in the holes so that the tips of your fingers touch the bottom of the jars.

"After you have your fingers in the water, I shall say a number of words, about one minute apart. After each word, you answer with the first word or

1. The apparatus was set up by Mr. Laffette, physiological assistant.

thought that comes to your mind. Remember, you say the first thing that comes to your mind, whether it makes sense or no, and do not move or take out your fingers, until I tell you to do so. Now, make yourself as comfortable as you can and put your fingers in the holes."

In the meantime, the assistant [2] has pulled down the window shade to darken the room. After the instructions, the subject puts his or her fingers in the jars, and the assistant fills in the name and age in the record which she holds, while the experimenter balances the bridge against the body resistance. When the bridge is balanced, the experimenter reads off the resistance in ohms to the assistant.

Then three practice words are given (book, sparrow, walk) for which no readings are taken. They are given in quicker sequence than the actual stimulus words, but are always preceded by a signal in the form of a statement: "Now, I am going to give you another word." This signal precedes all the stimuli.

After the practice words, the bridge is rebalanced and the experiment starts.

Practice Words	*Stimulus Words*		
Book	1. Mother	8. Tree	15. Sex
Sparrow	2. Grass	9. Spooning	16. White
Walk	3. Stink	10. Pavement	17. Love
	4. Chair	11. Man [3]	18. Stone
	5. Murder	12. String	19. Kiss
	6. Corner	13. Home	20. Pencil
	7. Woman	14. Glass	21. Marry

After the experiment the subject makes an introspective rating on the basis of I to II, inclusive, for the most affective words. To do this the subject is directed to sit at a table in the adjoining laboratory room where the temperature of the finger is again taken and then the instructions for the introspective rating are given as follows:

"I want you to pick out 11 words which are the most emotional for you from the list I gave you and rate them for me. Put the number '1' after the word that arouses the most emotion (gets you most excited or stirs up most feeling); '2' after the word with a little less feeling or emotion; '3' after the next, etc. Do this to and including '11.' You will then have 11 words more or less emotional, picked out and graded from '1' to '11,' '1' standing for the most emotional (or exciting) of the words you select, and '11' for the least emotional of the words you have chosen. If you are not satisfied with your first arrangement, you may change it."

For normal subjects, the first sentence alone of the instructions varies to read:

"We want you to help us make a test, which will be useful in our studies with the galvanometer." (Same as for patients after the first sentence).

Instructions for subsequent records are the same for normal and sick subjects and run as follows:

"We want you to take again the test you took last week in order to see how much better you are this week. You remember that all you have to do is to put the first finger of each hand in the holes so that the tips of your fingers

2. Lenore D. Jakob has been the assistant throughout the entire period of experimentation.

3. The stimulus word "man" is not used in the tables and charts that show the means for the grouped affective and nonaffective responses.

touch the bottom of the jars. Then remain as quiet as you can. When I say a word I want you to answer with the first word or thought that comes to your mind.

"Put your fingers into the holes and make yourself as comfortable as you can. Do not move or take out your fingers until I tell you to do so."

The results reported in this paper are based on thirteen normal male subjects, (first and second series), and forty-four male patients (second series). Of the normal subjects, three were physicians, five were psychologists, two were medical students and three were male nurses. Of the patients, twenty-three had dementia praecox and four paresis. The ages of the patients ranged from 15 to 77 years and those of the normal subjects from 21 to 43 years.

RESULTS AND COMMENTS

In view of the difficulty some workers have in devising a satisfactory set-up, it is interesting to note [4] that: The apparatus as described is sensitive enough to give fairly large deflection for comparatively mild affective and nonaffective stimuli. At the same time, it is not so sensitive that it will register the effects of the heart beat or respiration.

A slight deflection will occur when the subjects take a very deep breath or make a rather violent expiration. (That deep respiration has taken place is observed by the assistant and a notation made.) Seldom is the effect of such an inspiration or expiration superimposed on the response to the stimulus word.

The apparatus will not register slight movements of the head, body, hands, fingers and legs. When large or violent movements are made, a deflection occurs, but in these cases the movements are observable by the assistant, and the nature of the deflection can be recognized by the experimenter. Galvanic responses due to movements are immediate in initiation (practically no latency) and quick in their completion.

There is no constant drift of the beam of light due to polarization or to very small changes in the temperature of the solution. In experiments in which polarizable electrodes are used, accurate records are impossible.

In most cases, it is not necessary to rebalance the apparatus before the experiment is finished if accurate initial balancing has been made.

Temperature of the Solution.—It was discovered that the temperature of the zinc sulphate solution was of tremendous importance. Unquestionably, many of the irregularities and much lack of uniformity of results are due to the neglect of temperature standardization. (*a*) It is difficult to get a satisfactory record with the solution temperature lower than 28 C. In most cases when the temperature is below 28 C., the resistance is so high that the deflections are very slight or absent, or it is entirely impossible to balance the subject under 100,000 ohms.

4. Although the method of recording employed is accurate and practical, a photographic attachment would be an improvement. It would make for greater exactness and also give a record of the qualitative differences in the reaction.

(*b*) In the second series of records taken (most of the records are based on the second series), the maximum temperature was 34 C., the minimum 32 C., and the mean 32.1 C. for normal subjects. For patients the temperature of the solution varied from 29 to 34 C. The optimum temperature is from 31 to 33 C. (table 1).

The room temperature was recorded and considered in relation to the resistance of the subject and the temperature of the solution. It was found that when the room temperature was from 19 to 23 C., the best results were obtained. The maximum temperature was 22 and the minimum 16 C. for normal subjects. In the case of the patients, the room temperature ranged from 16 to 31 C. (table 1).

TABLE 1.—*Maximum, Minimum, Mean*

	Solution, Degrees C.		Hand, Degrees C.		Room, Degrees C.		Time, Minutes	Resistance
	Initial	End	Initial	End	Initial	End		
Normal Cases								
Maximum	34	33	33.9	35	22	23	25	70,000
Minimum	32	31	27.5	25	16	15	5	28,000
Mean	32.1	31.7	31.9	31	20	20.6	10.4	40,470
Dementia Praecox								
Maximum	34	33	37.1	35.6	31	31	35	90,000
Minimum	29	29	22	23	16	18	5	15,620
Mean	31.6	31.3	30	30	22.6	23.07	10.5	35,689
Manic Depressive Manic								
Maximum	34	34	37.2	35	30	30	20	52,000
Minimum	30	29	24.5	27	17	19	5	16,000
Mean	31.8	31.3	30	32	21.6	22.5	10.6	31,516
Manic Depressive Depressed								
Maximum	33	32	35	35	30	30	20	69,000
Minimum	30	29	25	25	19	20	10	17,000
Mean	31.3	30.3	31	31.5	22.9	23.2	13.1	37,392
General Paresis								
Maximum	33	32	34.4	33.9	26	26	20	48,000
Minimum	31	31	29	29	19	19	5	22,000
Mean	32	31.7	31.5	32	22.2	22.2	9.7	33,852

Resistance.—A record of the resistance of the subject as measured by the Wheatstone Bridge is significant, but has no value for differential diagnosis. (*a*) The mean resistance of normal males is 40,470 ohms, for manic patients 31,516 ohms, for depressed patients 37,392 ohms and for patients with dementia praecox 35,698 ohms (table 1). (*b*) The relationship between the amount of resistance and temperature of the solution, and between the resistance and the amount of deflection is important. If the temperature of the zinc sulphate solution decreases more than a degree, or goes below the lower limit of the optimum temperature, the resistance increases, and as the resistance increases the amount of deflection decreases. The inverse agreement between the resistance and deflection is only general and not in exact proportion as W. W. Smith claimed. It is apparent that a comparison

of cases is impossible without a correction on the basis of resistance, and that then the comparison can be made only with more, but not with absolute, certainty. It also demonstrates the uselessness of drawing conclusions of galvanic significance from differences in resistance without considering temperature factors. To claim that patients with dementia praecox have a greater resistance than manic-depressive patients is inaccurate if the temperature of the solution is not standardized.

(*c*) Another factor which must be considered is the differences in the temperature of the hands and fingers of different subjects. Some patients with dementia praecox have low body temperatures. When this is taken into account, in conjunction with the solution temperature, the greater body resistance of patients with dementia praecox disappears. For example, in the experiment reported it was found that a few subjects with dementia praecox had to rub their hands or hold loosely an electric bulb in order to be able to balance their resistance under 100,000 ohms. As a group, a smaller mean resistance was obtained in cases of dementia praecox than in subjects with manic-depressive (depressed) psychosis and in normal subjects (see table 1).

(*d*) There are no significant differences in resistance between patients and normal persons (table 1).

Length of Time Required in Taking an Individual Record, and Habituation.—(a) The mean time required to get a record of normal persons was 10.4 minutes, the minimum was 5 minutes, the maximum 25 minutes. The time required for patients varied from 5 to 35 minutes (table 1).

(*b*) The time element has meaning in connection with fatigue, restlessness and habituation.[5] If the experiment lasts long, the effects of all three factors increase.

(*c*) Some emotional habituation to this type of stimulation takes place as the experiment continues. In other words, if the stimulus words at the end of the list were given first, there would be a slightly greater deflection than when they are given at the end. However, on the basis of length of time consumed in each sitting, the amount of habituation is not great. A reversed order of presentation of stimuli was used in the second series of records without any noticeable changes in the relative amount of deflection. The habituation factor has little bearing on the results reported here because of the manner in which the stimuli and results are presented. First, comparisons can be made between the results of successive stimuli; second, when the deflections for the affective and nonaffective stimuli are taken together, the ratio

5. Wechsler calls it "exhaustion phenomenon."

between the two is not disturbed in any significant measure, because there are an equal number of affective and nonaffective stimuli in both halves of the list.

In comparing the amount of deflection for affective and nonaffective stimuli, it is well to keep in mind the fact that the differences might be greater if the affective and nonaffective words were given to the subjects in separate groups. There may remain a slight residue of emotivity when the nonaffective stimulus follows the affective word. The reasons for using the affective nonaffective order were to avoid the criticism of habituation and to prevent the accumulative effect of successive emotional stimuli.

Anticipatory Responses; Surprise, Tension-Set.—(*a*) A few writers claim that the galvanic response may be due in large measure to the set, tension or surprise accompanying the stimulus. With some patients, when no preparatory signal is given, there is some deflection due to the slight startling effect of the stimulus. If this deflection is superimposed on the psychogalvanic deflection related to the emotional or nonemotional stimulus, the quantitive measure is misleading. In such cases one would have a deflection for some stimuli which otherwise would give none. Giving a preparatory signal eliminates nearly all surprise deflections. In most cases there are no galvanic deflections to the preparatory stimulus. With subjects who give large psychogalvanic deflections there usually are small deflections (in relation to the size of the true deflection) following the signal. However, having given a response to the signal, the "tension-set" seems to have disappeared, producing no additional surprise deflection in conjunction with the response to the stimulus. When a surprise deflection precedes the true response the former can be recognized by its quickness of initiation and progression. The use of a preparatory signal eliminates it almost completely. This is shown by the large number of zero deflections. Another supporting point lies in the fact that the "tension-set" deflection is uniform for affective and nonaffective stimuli. It is practically a constant within each record except on very rare occasions when some new factor has entered to increase the "tension-set" preceding the next signal.

(*b*) On rare occasions an anticipatory response occurs which is similar to the "tension-set" deflection, except that the stimulus in those instances is central and not the preparatory signal or word stimulus given by the experimenter. The anticipatory responses die out quickly. When they occur just at the beginning of the stimulus, their effect is subtracted from the total deflection. The beginning of the true response is recognized by the acceleration taking place after a momentary break in the anticipatory response.

In view of the still meager knowledge of psychogalvanic phenomena, it is evident that the mathematical treatment of results, such as the employment of a mean, etc., is not satisfactory for diagnostic and clinical purposes. The most reliable and useful method is the study of individual cases. For that reason, tables of individual records will be presented and discussed in a later paper. However, in order to show that the galvanometer does measure differences in conductivity or body resistance or skin resistance under affective and nonaffective stimulation, the use of the mean is effective. If each response were studied, and affective and nonaffective stimuli grouped on the basis of individual associations and emotional coloring, the results would be more impressive. This is illustrated by the word "white." The word is com-

TABLE 2.—*Mean Galvanic Deflection for Ten Affective and Ten Nonaffective Stimuli of Classified Groups*

	Introspective				Test Arrangement			
	Noncorrected		Corrected		Noncorrected		Corrected	
	Affective	Non-affective	Affective	Non-affective	Affective	Non-affective	Affective	Non-affective
First series, 6 cases, normal males	8.25	4.02	27.61	15.05	8.25	4.02	27.87	14.80
Second series, 7 cases, normal males	5.01	2.43	19.60	8.27	5.36	2.08	18.42	6.90
First plus second series, 13 normal subjects	6.63	3.22	23.60	11.66	6.76	3.09	23.14	10.85
Dementia praecox (23 cases)	0.89	0.59	2.83	1.98	2.28	1.08	7.22	3.63
Manic-depressive manic (11 cases)	3.16	2.29	8.29	6.37	3.59	1.86	10.77	5.19
Manic-depressive depressed (6 cases)	4.06	2.57	10.92	6.70	4.85	2.38	14.21	6.90
Manic-depressive manic plus manic-depressive depressed (17 cases)	3.61	2.43	9.69	6.53	4.17	2.12	12.49	6.04
General paresis (4 cases)	0.4	0.06	1.52	0.23	0.78	0.13	2.90	0.47

paratively nonaffective for most people. Yet some subjects give the association "pure" and a large deflection. That deflection should, therefore, be counted with the affective group for those subjects. This cannot be done according to the test arrangement,[6] and introspectively (in most cases) the subject will not recognize "white" as an affective word. But even if one groups emotional and nonemotional stimuli on the basis of introspection (with its factors of dishonesty and inability to evaluate) or according to the test arrangement (based on the experimenter's evaluation), one still gets definite indications of marked differences in galvanic deflections. One might say no matter how the results are treated, there are unmistakable evidences of quantitative differences in response to affective and nonaffective stimuli.

6. This is amplified later in footnote 7.

As a rule, normal persons are more accurate in their introspections than patients. When introspection is more exact, the difference between the means for the affective and nonaffective group of stimuli is greater according to the introspective arrangement than according to the test arrangement.

The general thesis, "Does the galvanometer measure differences of emotional response?" can be approached from three angles.

(*a*) One method is the comparison of the average means of affective and nonaffective stimuli responses. Table 2, using the test arrangement—corrected means [7]—shows that all the normal subjects gave an average deflection of 23.14 cm. for the ten affective words and 10.85 cm. for the ten nonaffective words. Patients with dementia praecox gave an average deflection of 7.22 cm. for the affective and 3.62 cm. for the nonaffective; manic patients, 10.77 cm. and 5.19 cm.; depressed patients, 14.49 cm. and 6.90 cm.; paretic patients, 2.90 cm. and 0.47 cm. Chart 1 presents graphically the same facts for several groups. The response to affective words is shown by the dark column and the response to nonaffective words by the light column.

(*b*) Table 3 shows the relationship of the means for the emotional and nonemotional stimuli for each record of illustrative individual cases. Chart 2 shows the ratios between the means of the affective and nonaffective stimuli of the first five patients with dementia praecox and for the first five normal subjects used. Again the affective is represented by a dark and the nonaffective by a light column. That is the

7. In presenting the grouped results, I used two arrangements. One arrangement is on the basis of the subject's introspective rating, the other according to the test arrangement. The first is derived from the subject's choice of the eleven most emotional words from the list of twenty-one. If the word of the same sex (man) is included in the chosen list that word is excluded from the results, and there still remain ten affective words. Since the list is selected according to the relative affectivity of the words, the last word chosen can be excluded (if the same sex word was not selected) in cases of error, or included if one of the other words has been subject to error. In the test arrangement, as previously stated, the experimenter's estimate of affectivity determines the list of emotional or nonemotional stimuli. The test arrangement is on the basis of alternation of affective and nonaffective words, as indicated by the order of presentation. Under either method the mean is the average deflection of the ten emotional and ten nonemotional words in the list.

The noncorrected mean is the average deflection, regardless of differences in resistance. The corrected mean is derived by multiplying the average deflection for the ten affective and ten nonaffective words of each record for each patient by the resistance and then dividing the result by 10,000. The divisor 10,000 is used merely to reduce the size of the numerals designating the mean deflection. In cases in which a change in resistance had to be made in the course of taking a record, the mean resistance for that sitting was used as the multiplier, provided the change was made on the tenth or eleventh word.

second method of showing that the galvanometer measures differences of affectivity. Instead of including all the records, a descriptive statement covering all the records will demonstrate the point. Of the 44 patients and 13 normal persons, none has a lower mean for affective stimuli than nonaffective stimuli when the records of each subject are lumped together so that an average deflection for each case can be

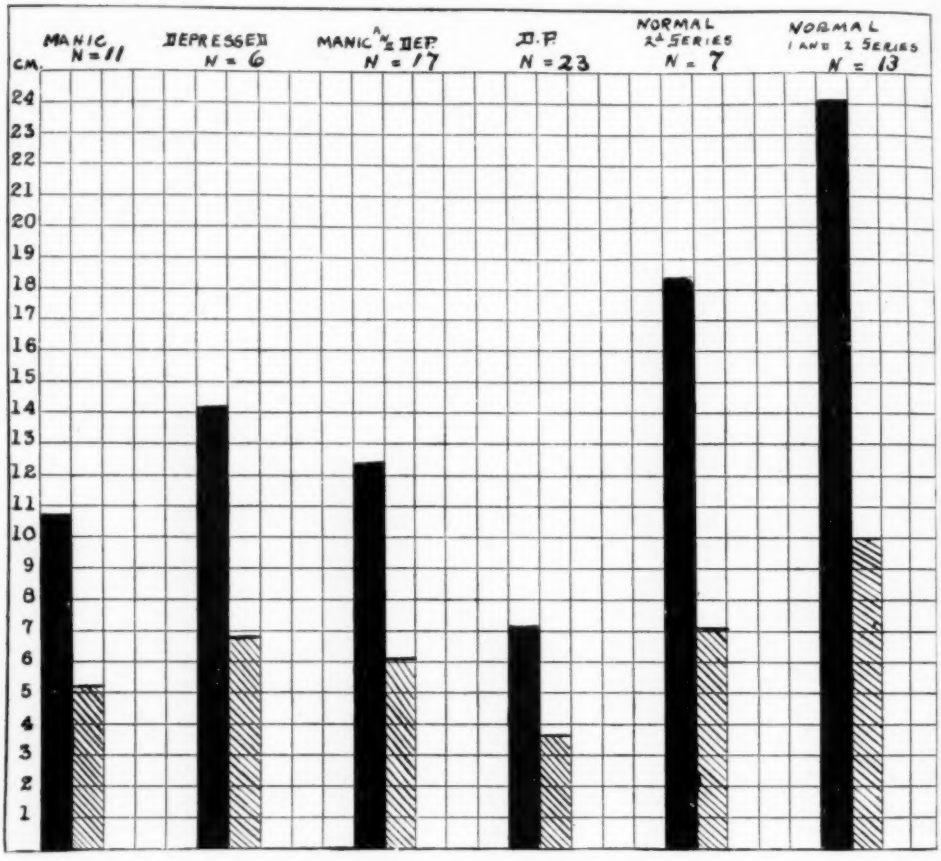

Chart 1.—Graphic representation of the mean galvanic deflection for ten affective and ten nonaffective stimuli of the classified groups. *N* represents the number of cases.

obtained. Of the 175 records used in the results, only 13 show a greater mean deflection for the ten nonaffective words over the affective words (according to the test arrangement). The introspective arrangement is less reliable because of the increasing lack of introspective ability as the mental condition becomes worse, and because of the emotional associations built around what are usually nonemotional

stimuli. For example, one hypomanic patient gave an unusually large deflection for glass, and explained that his free word association "table" was given because on the previous day he had been much upset by the breaking of a glass while moving a table. A depressed patient consistently gave a large deflection for glass, to which he responded with the word "tumbler." His history disclosed that he had swallowed pieces of glass which he had bitten from the top of a tumbler with

TABLE 3.—*Mean Galvanic Deflection for Each Record of Illustrative Individual Cases*

Type of Case	Date	Test Arrangement; Corrected Mean in Cm.	
		Affective	Nonaffective
Manic-depressive depressed	5/11/27	12.3	9.7
	5/18/27	20.0	23.0
	5/25/27	6.6	4.9
	6/ 8/27	12.4	7.5
	6/22/27	55.2	14.5
Manic-depressive depressed	5/18/27	13.5	2.6
	5/25/27	3.6	4.2
	6/15/27	19.9	17.8
	6/22/27	3.5	2.5
	6/29/27	0.4	1.1
	7/ 6/27	5.9	4.4
	7/13/27	2.4	1.0
	8/ 3/27	2.1	1.0
	9/14/27	15.3	3.3
Manic-depressive manic	9/ 6/27	9.4	6.9
	9/13/27	1.3	3.7
	9/20/27	5.0	1.6
	9/27/27	4.1	1.3
	10/ 3/27	4.0	0.4
	10/10/27	6.7	0.0
	10/17/27	3.3	0.7
	10/24/27	3.3	2.2
Dementia praecox	5/11/27	8.8	5.4
	5/18/27	11.0	4.1
	5/25/27	5.3	1.3
	6/ 8/27	14.6	11.0
	6/22/27	7.5	0.9
Dementia praecox	5/11/27	5.2	4.5
	5/18/27	0.5	0.4
	5/25/27	8.6	0.0
	6/15/27	10.6	3.4
	6/22/27	18.9	11.2
	6/29/27	1.5	1.5
Normal	9/20/27	49.5	19.3
	9/27/27	39.5	18.3
Normal	9/23/27	17.3	3.8
	10/30/27	6.29	0.0

suicidal intent. That may readily be the explanation of the large deflection. Many such situations emphasize the fact that the galvanometer shows affective variations, for in spite of the inclusion of these larger individual deflections for the nonemotional words in the group of nonaffective stimuli, one still gets approximately double the mean deflection for the affective words over the nonaffective.

Illustrations like those cited demonstrate the need for studying each response of each case individually and evaluating the affectivity on such

a study, if the results of the galvanometer are to be used diagnostically and accurately. The interpretations should be made on the basis of the associated word response (and what it might disclose by probing), on the history of the case and on what a discussion of the introspections will indicate.

(*c*) A third way to demonstrate the effectiveness of the galvanometer for measurement of emotional reactions is through the use of tables showing the individual deflections for each stimulus at each sitting and the mean deflection for each stimulus word. Since this paper is not intended to cover individual case studies, tables showing the individual responses are not included. Table 4 shows the mean deflection for each stimulus word of the various groups studied. It should be noted that table 4 gives the means of actual deflections without any corrections. To compare the groups, a correction for each galvanic deflection of each case

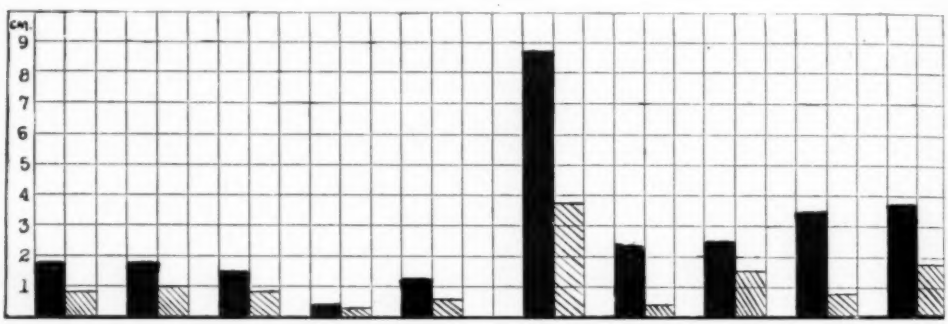

Chart 2.—Mean deflections for affective and nonaffective words of the first five dementia praecox and the first five normal subjects used in the experiment.

should be made. The purpose of the table, as it now stands, is to show the see-saw effect of the alternating affective and nonaffective stimulation. The ratios between the affective and the nonaffective stimulus responses are fairly exact, because the resistance throughout a record varies little except in a few cases. It is doubtful if any significant variations would occur between alternating emotional and nonemotional stimuli and their means as they now stand and as they would be if corrected.

Table 5 gives the rank order of mean galvanic deflections for all stimulus words for normal subjects and patients. Were the results corrected, there might be slight changes in the amount of deflection and the order of affectivity. There would not be any large change in order because of the fact previously stressed that in each individual record the relation of one deflection to another is uniform. A study of table 5 suggests several interesting facts. The stimulus words which

were selected as affectively toned gave larger deflections than the nonaffective words in the case of normal subjects and patients. There is one exception in the case of the word stimulus "glass." It had been used as a nonaffective word, but it gave a fairly large deflection in the

TABLE 4.—*Mean Galvanic Deflection for Each Word of Classified Groups*

	Normal, Second Series	Dementia Praecox	Manic-Depressive Manic	Manic-Depressive Depressed	General Paresis
Mother	5.19	2.45	4.52	4.62	1.13
Grass	2.62	1.63	2.04	3.29	0.0
Stink	4.71	1.56	1.92	4.39	1.27
Chair	2.03	1.35	2.31	2.43	0.12
Murder	3.37	1.51	3.09	5.23	1.50
Corner	2.42	1.45	1.98	2.79	0.31
Woman	3.00	2.02	2.87	4.65	0.34
Tree	2.36	1.30	1.45	1.77	0.06
Spooning	6.23	2.70	3.50	5.56	0.75
Pavement	1.87	0.81	1.41	2.13	0.00
Man	3.30	1.26	1.51	4.24	1.00
String	2.20	1.25	1.90	1.55	0.06
Home	5.07	1.92	2.65	2.76	0.62
Glass	1.37	1.23	3.05	4.96	0.31
Sex	6.30	2.51	4.58	6.23	0.52
White	2.70	0.69	1.70	3.84	0.22
Love	5.01	1.90	3.81	5.85	0.50
Stone	1.70	0.87	1.56	2.39	0.20
Kiss	5.12	2.87	4.97	5.22	0.50
Pencil	1.37	0.80	1.15	1.29	0.07
Marry	7.28	1.56	2.86	4.92	0.87

TABLE 5.—*Rank Order of Stimulus Words According to the Mean Galvanic Deflection*

Seven Normal Persons				Forty-Four Patients			
Rank Order	Order of Presentation	Word	Mean Galvanic Deflection	Rank Order	Order of Presentation	Word	Mean Galvanic Deflection
1	21	Marry	7.28	1	15	Sex	3.46
2	15	Sex	6.30	2	19	Kiss	3.36
3	9	Spooning	6.23	3	1	Mother	3.18
4	1	Mother	5.19	4	9	Spooning	3.07
5	19	Kiss	5.12	5	17	Love	3.01
6	13	Home	5.07	6	5	Murder	2.83
7	17	Love	5.01	7	21	Marry	2.55
8	3	Stink	4.71	8	7	Woman	2.46
9	5	Murder	3.37	9	14	Glass	2.38
10	11	Man	3.30	10	3	Stink	2.28
11	7	Woman	3.00	11	11	Man	2.00
12	16	White	2.70	12	13	Home	1.98
13	2	Grass	2.62	13	2	Grass	1.76
14	6	Corner	2.42	14	6	Corner	1.63
15	8	Tree	2.36	15	16	White	1.61
16	12	String	2.20	16	4	Chair	1.55
17	4	Chair	2.03	17	18	Stone	1.20
18	10	Pavement	1.87	18	12	String	1.19
19	18	Stone	1.70	19	8	Tree	1.14
20	14	Glass	1.37	20	10	Pavement	1.08
21	20	Pencil	1.37	21	20	Pencil	0.85

case of several subjects composing the abnormal group. The large responses for glass which several patients gave, as previously described, explain this reversal in order. In my first series, "sex" gave the greatest mean deflection, in the second series "marry" apparently was the most affective, with "sex" a close second. In general, it is safe to conclude that stimuli related to sex experience give the

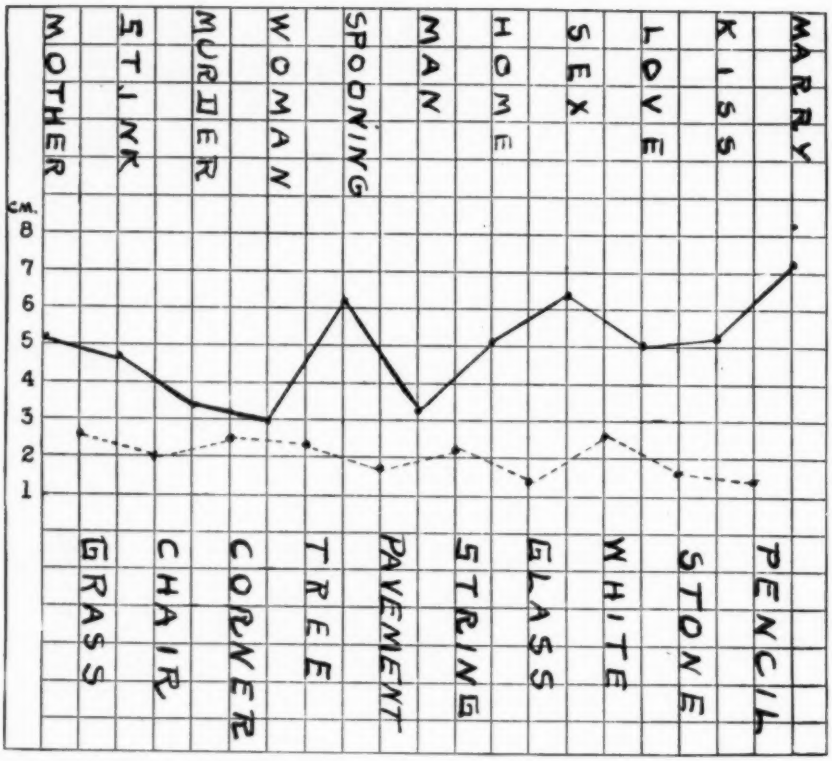

Chart 3.—Normal subjects; graphic representation of the mean galvanic deflections for affective and nonaffective stimuli in thirteen cases. The heavy line represents the affective, the broken line, the nonaffective words.

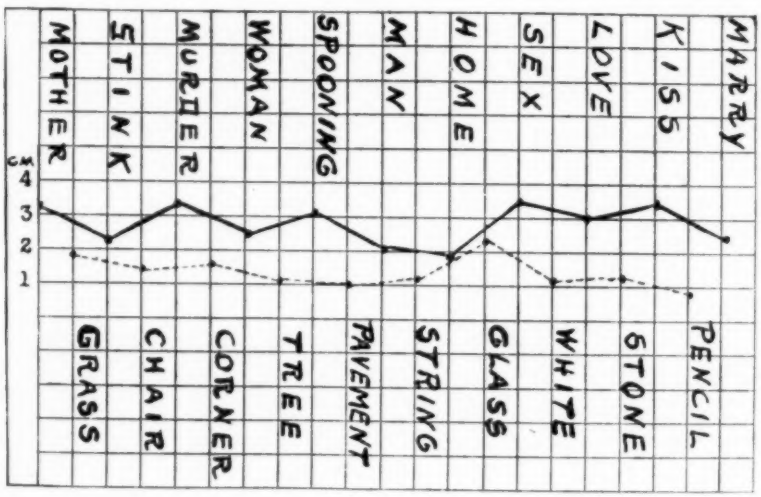

Chart 4.—All patients; graphic representation of the mean galvanic deflections for affective and nonaffective stimuli in forty-four cases. The heavy line represents the affective, the broken line, the nonaffective words.

greater deflections except in cases in which unique, strong associations have been built up for particular situations. The first series included four female patients and two normal women. There were not enough female patients to enable me to make any comparisons between the sexes. There was a suggestive tendency that "sex" has more emotional meaning for males than females and that "love," with less sex meaning, seems to have greater affectivity for female subjects.

Charts 3 and 4 show graphically the differences in the mean deflections between the affective and nonaffective stimuli. The solid line represents the emotional words and the broken line the nonemotional words, according to the experimenter's evaluation (test arrangement). Each connected point represents the mean galvanic deflection for each stimulus in their order of presentation. Stimulus word 1 is at the extreme left, solid line, and word 21 is at the extreme right, solid line. The solid line has the first and last word, because the first and last are affective words. To illustrate, the first point on the solid line is the mean deflection to "mother," the first point on the broken line is "grass," second point, solid line, is "stink," and second point, broken line, is "chair," etc.

CONCLUSIONS

1. The psychogalvanic reflex is related, either directly or indirectly, to affective responses.

2. The galvanometer lends itself in a most promising manner to an investigation of the emotions.

3. To get dependable results the conditions of the experiment must be standardized as to: (*a*) room temperature, (*b*) solution temperature (if wet electrodes are used), (*c*) electrodes, nonpolarizable, and with uniformity of contact, and (*d*) finger temperature (if contacts are made there).

4. The psychogalvanometer opens the way to a study of the affective phase of different psychoses and gives promise of providing knowledge useful in diagnosis and treatment. For interpretative and diagnostic purposes each case must be studied individually and separately for each response.

5. The results indicate that when conditions are standardized as previously cited:

(*a*) There are no significant differences in resistance for the various groups of patients and normal subjects.

(*b*) The resistance increases in most cases as the experiment continues.

(*c*) With marked increase in resistance there is a decrease in deflection and vice versa—but not in direct proportion.

(*d*) A stimulus usually causes a decrease in resistance (three exceptions in 188 cases). After the deflection, the beam of light, on its return, usually goes beyond the starting point, indicating an accumulative and gradual increase in resistance as noted under (*b*).

(*e*) If liquid electrodes of zinc sulphate are used there is an optimum temperature, probably somewhat dependent on the set-up used.

(*f*) A galvanic deflection to large bodily movements is recognizable by its almost immediate initiation and rapidity of completion. It is in the same direction as affective responses.

(*g*) Some habituation phenomena (e. g., decrease in deflections for repeated stimulations) take place in normal subjects and slightly in patients whose condition has not changed. If the mental condition has grown worse the habituation phenomenon is augmented by decreased emotivity, and if the mental condition has improved, the habituation phenomenon is negated by the increase in emotivity.

(*h*) Tension and anticipation appear to be factors only in so far as they induce affective states.

EPIDEMIC ENCEPHALITIS WITH CEREBELLAR SYMPTOMS

A CLINICAL STUDY OF TWO CASES *

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Although numerous cases of epidemic encephalitis with involvement of the vestibular connections have been reported in the literature, a predominance of cerebellar symptomatology is less frequently found. When cerebellar manifestations have been described they have, in most cases, been overshadowed by the parkinsonian syndrome.

REPORT OF CASES

In view of the comparatively rare incidence of cerebellar symptomatology in chronic epidemic encephalitis, the following cases are presented.

CASE 1.—*History.*—B. B., a high school student, aged 18, was admitted to the University Hospital on April 25, 1928, complaining of headache, dizziness and staggering. The symptoms had begun one year previously when he noticed blurred vision and diplopia on continued reading. This difficulty increased in severity during the next few weeks. There were no chills or fever, but a dull pain between the shoulders and a persistent headache were present and at times became agonizing. Seven months after the appearance of diplopia, there developed moderate photophobia, dizziness when standing or walking, and an unsteady gait with staggering, usually to the left. Nausea and projectile vomiting occurred at weekly intervals, the latter not synchronous with the dizziness. He would often fall asleep for long periods, being awakened with difficulty. Slight exertion caused profuse sweating, but there was no general weakness. Lacrimation was increased.

Previous to the onset of the illness, he had been subject to frequent colds. Measles, diphtheria and two attacks of pneumonia occurred in childhood. He gave a history of trauma at the age of 12, resulting, so he was told, in a rupture of the liver. Recovery followed operation. At no time was there any illness resembling acute epidemic encephalitis.

Examination.—The general physical examination showed no pathologic condition other than diseased tonsils, enlarged cervical glands and some tenderness over the lumbar region. The blood pressure was 118 systolic and 80 diastolic.

There was no motor weakness or rigidity. The tendon reflexes were normal; Babinski's sign was not elicited. There was no impairment of touch, pressure,

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* Presented through the courtesy of Dr. William G. Spiller. From the Department of Neurology of the University of Pennsylvania.

pain, temperature, vibration, position or stereognostic sensibility. There were fine tremors of the eyelids and tongue and a tremor of the left hand slightly suggestive of parkinsonism. In walking, the patient did not swing his arms as much as a normal person.

Vision was 6/5 in both eyes, and the visual fields were not contracted. The media were clear; the disks were round and of good color, with no choking. The pupils responded to light and in accommodation. Convergence was definitely weak (fig. 1). The patient showed about 3 degrees of esophoria for 6 meters, but a high exophoria for near vision. There was no nystagmus. Aside from the defective convergence, the extra-ocular movements were full and equal in all directions. The fifth and seventh pairs were normal. The audiometer test showed a loss in

Fig. 1 (case 1).—Weakness of convergence.

hearing of only 5.8 per cent on the right and 2.7 per cent on the left. A vestibular examination by caloric and turning tests showed that the vestibular pathways were normal.

Cerebellar Symptoms.—In standing erect or in walking, the patient consistently staggered to the left and backward, in spite of a wide base. This occurred to a greater extent with the eyes closed, but was almost as pronounced with the eyes open. When he attempted to rise from the supine position without the use of the arms, the feet were raised from the floor, the left higher than the right (fig. 2). He did not flex the knees in bending backward and would fall if not supported. When he brought the arms downward from a vertical extended position to a horizontal plane, with the eyes closed, the left arm was held at a higher level than the right, the fingers being flexed and separated (fig. 3). With the arms extended horizontally and with the eyes closed, pronation caused the left hand to be held higher than the right. There was moderate hypotonia in the left upper extremity, and the Gordon Holmes rebound phenomenon was present. The finger-to-nose,

finger-to-finger and heel-to-knee tests were well performed. Rapid pronation and supination of the hands on the knees revealed no adiadokokinesis.

Laboratory Tests.—Wassermann tests of the blood and spinal fluid were negative with all antigens. The spinal fluid was under a pressure of 4 mm. of mercury; it was clear and colorless, with a cell count of 2 (lymphocytes); the amounts of sugar and globulin contained were normal; the total protein was 1.7; the colloidal gold curve was 0000000000.

Course.—The patient was in the hospital for two months, during which he had constant headache, drowsiness and frequent attacks of projectile vomiting. The

Fig. 2 (case 1).—Dyssynergia on attempt to rise from supine position.

Fig. 3 (case 1).—Dyssynergia on bringing the arms from a vertical to a horizontal plane.

pulse and temperature remained within normal limits. The cerebellar signs remained constant, and he was discharged unimproved.

Comment.—The drowsiness, headache, diplopia, impaired convergence, and photophobia make a diagnosis of epidemic encephalitis most probable, even though a history of an acute attack is lacking, as is not infrequently the case. The absence of positive signs in the eyegrounds or of increase in the spinal fluid pressure make the diagnosis of

cerebellar tumor questionable. It is believed that the cerebellar gait, staggering, trunkal asynergia, hypotonia and dysmetria in the extremities of the left side justify the conclusion that the disease is of the cerebellar type, the vermis and left hemisphere being chiefly affected.

CASE 2.—*History.*—J. K., a white man,[1] a mill worker, aged 56, entered the University Hospital on Nov. 30, 1925, having been referred by Dr. A. M. Ornsteen, with a complaint of dizziness and staggering. The onset of the disease had occurred nine months previously, with a sudden attack of intense dizziness in which he fell to the floor. He felt a sinking sensation in the abdomen and vomited. For six weeks following this, he was delirious. A diagnosis of epidemic encephalitis was made by attending physicians at this time. In the period from the seventh to the eleventh week he had diplopia, staggering and hypersomnia in addition to vertigo and frequent vomiting. The diplopia and vomiting disappeared by the end of six months, but the other symptoms persisted. There had been no previous illness except sciatica in 1913.

Examination.—The patient was well developed and showed marked incoordination. The head, neck, heart, lungs and abdomen showed no abnormalities.

The tendon reflexes were normal, and no pathologic reflexes were elicited. Touch, position, vibration, pain and temperature sensibilities were normal. There was no diminution in motor power. The cranial nerves were entirely normal. Vision was good; the fields showed no cutting; the pupils reacted to light and in accommodation, and the extra-ocular movements were full. A Bárány test was not made.

The patient's gait was that of a drunken person. He bent forward at the waist and staggered greatly. The right arm was swung to a less degree than the left in walking. When he bent backward, the knees were not flexed until near the completion of the movement. A tremor of the left forearm and hand and of the upper left half of the trunk was present. Dyssynergia and adiadokokinesis appeared in the left upper extremity, though it was difficult to determine any increase in dysmetria at the initiation of any movement. The finger-to-nose test was accomplished much better with the right than with the left hand. Lying on his back on the floor, with the arms folded across the chest, he was unable to rise, as the feet came up as high as his head at every attempt. In addition to the observations noted, he presented to a remarkable degree the phenomenon of cerebellar catalepsy, being able to maintain a position on his back with the legs elevated from the floor for long periods.

Comment.—The diagnosis in case 2 may be questioned in view of the sudden onset, which is suggestive of a vascular lesion; this, however, is known to occur in acute epidemic encephalitis. The subsequent course of the disease, with delirium, persistent vomiting, diplopia and hypersomnia are more suggestive of encephalitis. The cerebellar signs were more pronounced than in case 1, and although there may have been simultaneous involvement of the vestibular pathways, the latter could not have been the predominant lesion.

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1. This patient was not seen by the author but is reported from the notes of Dr. William G. Spiller.

COMMENT

Wimmer [2] stated that in a few cases he has observed symptoms that might have been of cerebellar origin, but that a systematic examination of cerebellar functions never reveals typical disorders. He mentioned tremors and certain forms of torsion spasm as possibly of cerebellar origin, and quoted a case in which these occurred. He expressed the belief that adiadokokinesis in patients with the parkinsonian syndrome is a pseudophenomenon due to the rigidity and bradykinesia.

Barré and Reys [3] found cerebellar symptoms rare, having observed them in one case in a pure form and in another in association with myoclonic movements, although they considered the latter a doubtful case. Courtois-Suffit [4] and others described a cerebello-vestibular syndrome due either to encephalitis or to multiple sclerosis. Their patient had a tendency to cerebellar catalepsy and lateropulsion, but the vestibular symptoms predominated. Schaffer [5] and his co-workers reported a cerebellar syndrome with absent tendon reflexes due to epidemic encephalitis. Dysmetria, adiadokokinesis, hypotonia and cerebellar catalepsy were manifested. In a series of fifty cases Heagey [6] found eight with cerebellar manifestations. In only three of the eight was there a predominance of the cerebellar elements. All three showed nystagmus and ataxia. Two patients complained of falling, and one fell to the same side on which a lesion was demonstrated at necropsy. In one case, the dentate nucleus showed gross changes.

Kawata [7] described cases in which he found changes in the cerebellum as the result of epidemic encephalitis. It is interesting that these cases do not seem to have been chosen because they presented cerebellar symptoms. Kawata found these changes in cases in which the clinical manifestation was parkinsonism. He found the nucleus dentatus especially affected, in some cases showing a pronounced loss of cells. There were changes in the condition of the Purkinje cells similar to those occurring in these cells in senile conditions. Atrophy of the granular layer also occurred. He studied, in all, eight cases of parkinsonism in which he investigated especially the cerebellum, and in all eight he found all parts of the cerebellum affected and the meninges comparatively slightly involved. He considered it striking that the cerebellar changes have been overlooked by different investigators, except by Hohman and

2. Wimmer, August: Chronic Epidemic Encephalitis, London, Heinemann, 1924, pp. 352.

3. Barré and Reys: Bull. méd., Paris **35**:356, 1921.

4. Courtois-Suffit; Schaffer, H., and Garnier: Soc. de neurol., Paris, 1925, vol. 1, p. 441.

5. Schaffer; Celice; Seligmann, and Garnier: Rev. neurol. **2**:580, 1923.

6. Heagey, Francis, W.: New York M. J. & Rec. **116**:321, 1922.

7. Kawata, Akira: Arb. a .d. neurol. Inst. a. d. Wien. Univ. **29**:226, 1927.

Koenig, because the changes that he found were distinct and were present in young as well as in old persons. Levy also described late changes in the cerebellum in encephalitis.

It would seem probable that in those cases of epidemic encephalitis in which cerebellar symptoms have predominated, the pathologic changes would be even more prominent than were those described by Kawata.

SUMMARY

1. Two cases of chronic epidemic encephalitis with a predominance of cerebellar symptoms are described.

2. Five cases in which the cerebellar syndrome is manifested in encephalitis are mentioned in the literature. Seven other cases showing merely isolated cerebellar signs occur in the literature. Cell changes in all parts of the cerebellum have been found at necropsy in cases of encephalitis in which cerebellar symptoms did not occur or were not prominent.

BLASTOMYCOTIC MENINGO-ENCEPHALITIS

REPORT OF A CASE *

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Blastomycosis is caused by a group of allied yeastlike organisms, which are usually surrounded by a thick hyaline capsule and which multiply in the tissues by budding. Because of the latter characteristic, they have been called blastomycetes. The first case of blastomycosis was reported by Busse in 1894, and this was followed, in 1896, by the first study from the United States by Gilchrist and Stokes.[1] Since then numerous cases of the disease have been reported from various parts of this country. The larger proportion of the cases of blastomycosis studied from the clinical as well as the histologic and cultural standpoints, however, have been reported from Chicago.

Clinically, blastomycosis appears in cutaneous forms and as the so-called systemic blastomycosis. The cutaneous form is no longer regarded as uncommon, but systemic or generalized blastomycosis is still recognized as rare. The organisms in systemic blastomycosis have been recovered from the blood, and they are present in abscesses. They are found frequently in the sputum and occasionally in the urine and feces. Every organ may be affected, but the brain and particularly the meninges are rarely involved by the infectious process. The lesions of the brain are usually abscesses, seated in the deeper tissues, but Le Count and Meyers [2] reported a large conglomerate blastomycotic nodule, 4.5 by 3.8 cm. in size, in the cerebellum, resembling a solitary tubercle. Necrotic foci of the bones of the skull are one source of infection of the meninges.

A few years ago Wilhelmj [3] collected reports of six cases of blastomycotic meningitis, together with his own, in which no blastomycotic lesions could be demonstrated clinically in any part of the body. In two of these cases, autopsy showed no involvement of other organs. He suggested that it is probable that, when the meninges are infected during

* Submitted for publication, Feb. 18, 1929.

* From the Laboratory of the Rochester General Hospital.

1. Gilchrist, T. C., and Stokes, W. R.: The Presence of an Oidium in the Tissues of a Case of Pseudo Lupus Vulgaris, Bull. Johns Hopkins Hosp. **7**:129, 1896.

2. Le Count, E. R., and Meyers, J.: Systemic Blastomycosis, J. Infect. Dis. **4**:187, 1907. Le Count, E. R.: Blastomycotic Lesions of the Brain, J. Nerv. & Ment. Dis. **36**:144, 1909.

3. Wilhelmj, C. M.: Primary Meningeal Form of Systemic Blastomycosis, Am. J. M. Sc. **169**:712, 1925.

the initial period of invasion, death usually occurs before the general metastatic foci appear. From a clinical point of view, he considered these cases as a primary meningeal form of systemic blastomycosis, which may occur without gross clinical evidence of systemic invasion.

Various opinions exist regarding the histology of blastomycotic lesions. One opinion holds that lesions can be produced by the blastomycetes which cannot be distinguished histologically from those caused by tubercle bacilli, except by the recognition of the specific parasite. According to another opinion, however, the histologic picture of the lesions caused by these organisms resembles that of tuberculosis only in certain general points. The following case, which presents a chronic blastomycotic meningitis invading the brain tissue proper, is reported because of several interesting features. The lesion is a tumor-like mass, showing characteristics which are not entirely similar, histologically, to those found in tuberculosis. I have found constant characteristics which will differentiate them from a tubercle, without the demonstration of the organisms. These differences had already been noted by other observers, but not in such a long-standing process in the meninges. The case belongs clinically to the primary meningeal form and is so far as I know, the first case of systemic blastomycosis reported from New York State.

REPORT OF CASE

Clinical History.—F. H., a white man, aged 52, a blacksmith, had had occasional boils, but no serious skin trouble. The present illness began at the end of September, 1927, with pain in the head and neck. He continued to work until the first days of November, when the pain so increased that he could not endure it. During this time he had attacks of dizziness. He was first admitted to the Rochester General Hospital on the neurosurgical service of Dr. H. W. Williams on Nov. 12, 1927.

Physical Examination.—The patient had tenderness over the occipital region. There was a horizontal and vertical nystagmus with extreme lateral or upward motions of the eyeballs without strabismus. There was a slight choking of the disks. The upper extremities were somewhat rigid. Walking was difficult, and he acted as if he were about to fall at any moment. He had no Romberg sign.

Course.—The temperature was always normal except on one or two occasions, when it was 101 F. Laboratory observations: Nov. 16, 1927, x-ray plates of the skull were normal. The spinal fluid was under a pressure of 30 mm. of mercury, and contained 35 cells per cubic millimeter and a marked increase in globulin. The red blood cells numbered 4,950,000; white blood cells, 6,800; hemoglobin, 95 per cent. A differential count showed neutrophils 76 per cent; lymphocytes 24 per cent. The nonprotein nitrogen of the blood was 31.5 mg.

The symptom-complex which the patient presented was that of high intracranial pressure. It was considered to be due probably to a neoplasm, but the neurologic signs were confusing and rather meager in view of the prominence of the pressure signs. In order to obtain data for localization, a ventriculogram was attempted. On November 18, with the patient under local anesthesia, two burr holes were made in the occipital region, but only a few drops of fluid were

obtained from the posterior horn of either ventricle. Small pieces of brain tissue removed with the needle from deep in the occipital lobes failed to reveal any microscopic changes.

Following this, the symptoms increased in intensity and the edema of the disks increased to 3 diopters in each eye. As all attempts to localize the lesions had failed, a right subtemporal decompression was done on December 3, by Dr. H. W. Williams. With the patient under local anesthesia, a bone defect, 5 by 6 cm., was made in the temporal bone. The dura was tense, and the brain bulged outward but did not rupture. The cortex did not appear grossly abnormal. A great deal of trouble was experienced during this procedure from bleeding of a distinctly pathologic type from every point in the operative field. Not even the smallest vessels could be controlled without ligature. The blood coagulation time, taken after the operation, was thirteen minutes.

After decompression, the choked disks subsided to 1 diopter in each eye, and the other symptoms diminished in intensity. The patient was discharged on Dec. 10, 1927, considerably improved.

Interval History.—During the interval the headache was about as severe as before, and occipital in type. There was no vomiting. Vision was somewhat worse, and at times he saw double. The patient thought that he had some impairment of memory. He could not walk alone. On Dec. 22, 1927, he had a jacksonian convulsion beginning in the right arm and extending to the face and leg. He became unconscious. Following this he exhibited great difficulty in speaking for about two hours. He had four more similar convulsions, each followed by temporary aphasia, before readmission to the hospital on Feb. 15, 1928.

Second Admission.—Examination on readmission showed no new symptoms with the exception of a slight exaggeration of the deep reflexes on the right side. There was also at times some aphasia of a motor type and greater choking of the disks than previously. Babinski and Oppenheim signs were positive on the left side.

From these observations and the continued absence of parietal lobe symptoms it seemed fair to assume that there was a neoplasm in the left frontal region encroaching on the precentral area. The blood coagulation time had returned to normal.

On February 20 with the patient under colonic ether and local anesthesia, a large flap was turned down by Dr. Williams, exposing the rolandic and postfrontal areas. On opening the dura, an encapsulated neoplasm was found over the rolandic area extending backward under the skull over the parietal lobe. It was firmly adherent to the dura, but macroscopically was not attached to the cortex. A considerable area of bone had to be removed in order to reach the posterior edge. At the angle between the falx and the dura it disappeared in the direction of the corpus callosum. Its inner margin could not be reached, and it was transected at that point, a portion of the tumor attached to the falx being left. The brain was obviously still under considerable pressure after the tumor was enucleated and some difficulty was experienced in closing the dura. The bone flap was replaced and the scalp closed without drainage.

After the operation, the patient had a complete paralysis of the right leg and arm, with aphasia. This improved rapidly. He then had several convulsive seizures, both on the same and on the other side. Following this, the condition became rapidly worse. The temperature was usually from 100 to 102 F. in the evenings, but was 103 F. on March 20, 1928. A blood count on February 29 revealed: red cells, 3,910,000; white cells, 9,200; neutrophils, 78 per cent;

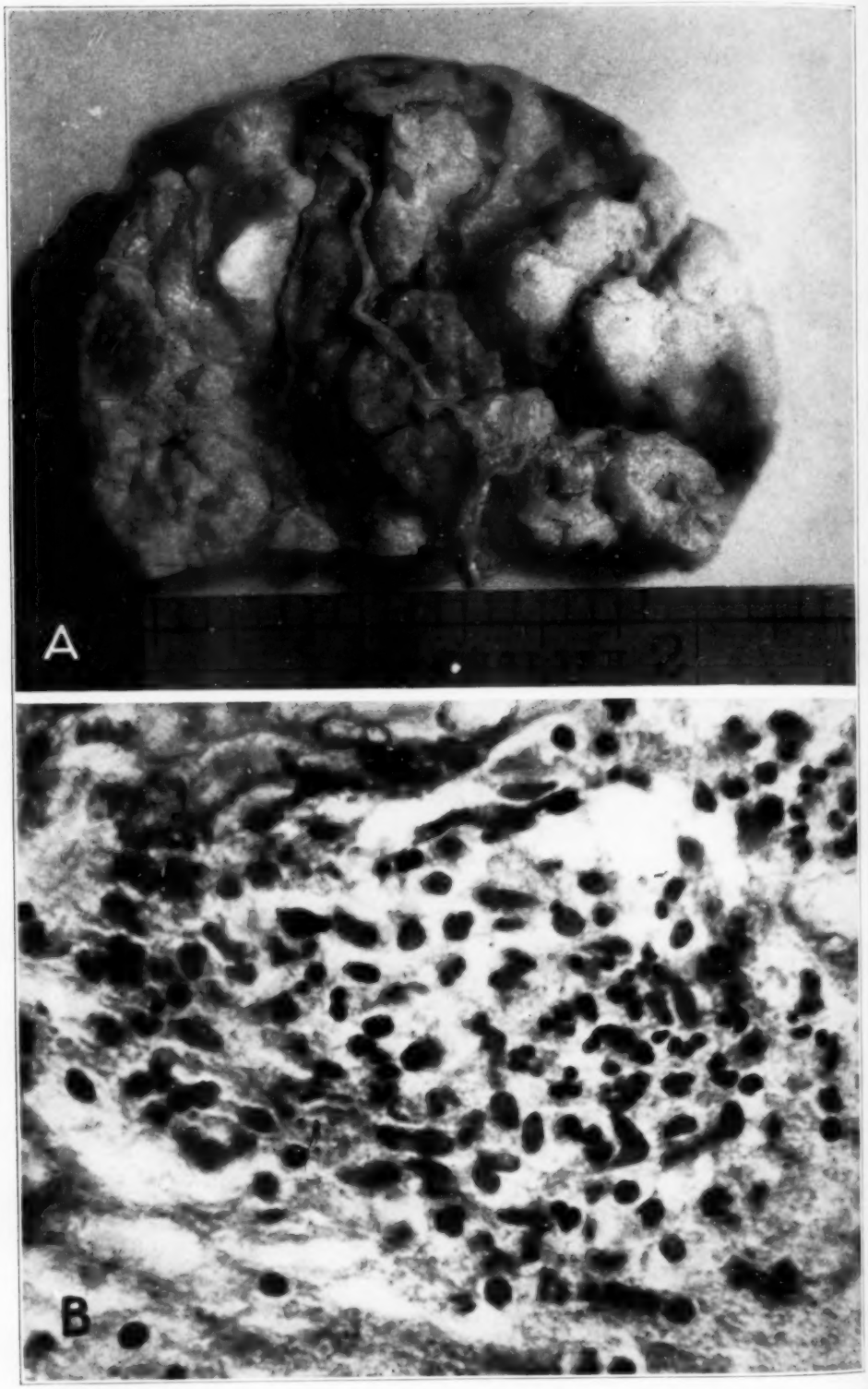


Fig. 1.—*A*, gross appearance of the granulomatous tumor; *B*, small abscess, representing the first histologic changes caused by the blastomyces.

eosinophils, 2 per cent, and lymphocytes, 20 per cent; hemoglobin, 77 per cent. The nonprotein nitrogen was 60 mg., and chlorides 540 mg. The patient left the hospital and died a few weeks later. No autopsy was obtained.

Gross Pathologic Changes in Tumor.—The tumor was fairly round, 6.5 cm. in diameter and 2 cm. in thickness (fig. 1 *A*). The thickest part was near the center; the edges were thinner, and here the two membranes of the pia-arachnoid could be definitely distinguished. The upper surface was fairly smooth, and the under surface was covered with rough granulations varying in size from that of a pinhead to that of a bean. Larger and small vessels passed through this surface; the cut section revealed grayish-white and irregularly scattered yellowish-white areas of varying size.

Histologic Examination.—Microscopically, three layers could be distinguished roughly on the cut surface of this tumor: an external layer just under the dura, an intermediate and an internal, which infiltrated the brain tissue.

The internal layer was formed by smaller and larger foci, irregularly infiltrating the brain substance. The foci consisted of neutrophil and eosinophil leukocytes. Among these cells could usually be found one, two or more spherical bodies of varying size (from 10 to 25 microns) surrounded by a hyaline capsule. Thus, the foci were miliary abscesses. Larger abscesses were also found, and in these part of the leukocytes were usually necrotic, their bodies lying among normal polymorphonuclear cells. These larger abscesses usually contained numerous parasites. Some foci were sharply outlined, while others showed an indefinite border. These abscesses represented the more recent lesions (fig. 1 *B*). The surrounding brain tissue was at first free from any reaction, but subsequently several blood vessels appeared, many of them growing in from the meninges. The adjoining connective tissue enclosed and practically encapsulated the abscesses (fig. 2 *A*), and some fibroblasts also grew in between the leukocytes (fig. 2 *B*). Giant cells were formed among the enclosing fibroblasts. These in the early stages often appeared of half-moon or horseshoe shape and contained the spherical bodies in varying numbers (fig. 3 *A*). Such foci represented older lesions. The connective tissue around them became more and more hyaline, and there were numerous plasma cells and fewer lymphocytes and neutrophil and eosinophil leukocytes in the wide tissue spaces. It is important to note that these cells did not show a definite ringlike arrangement around the foci. Only a few cells of this type could be found even in their immediate vicinity. The number of the giant cells may increase in the foci and their shape may resemble the Langhans type.

There were numerous necrotic areas in the intermediate layer of this tumor. The necrosis occurred mostly in the individual foci, and the neighboring hyaline connective tissue preserved its structure well. The vicinity of the necrotic areas was not characteristic. They were surrounded often by a hyaline connective tissue, in which there were only a few plasma cells and leukocytes. Near the edges of the necrotic areas were a few foci which could be considered as the oldest. They consisted of one or two giant cells enclosing spherical bodies, embedded in hyaline connective tissue, and a few leukocytes and plasma cells were also nearly always present. Occasionally, I noted blood vessels filled with red corpuscles in the center of the necrotic areas.

The external layer of the tumor was histologically similar to the internal layer, but the most recent pathologic changes, the abscesses, were missing. The hyaline connective tissue was abundant and was infiltrated with plasma cells. In this tissue were embedded fairly round foci containing leukocytes, fibroblasts and foreign-body giant cells.

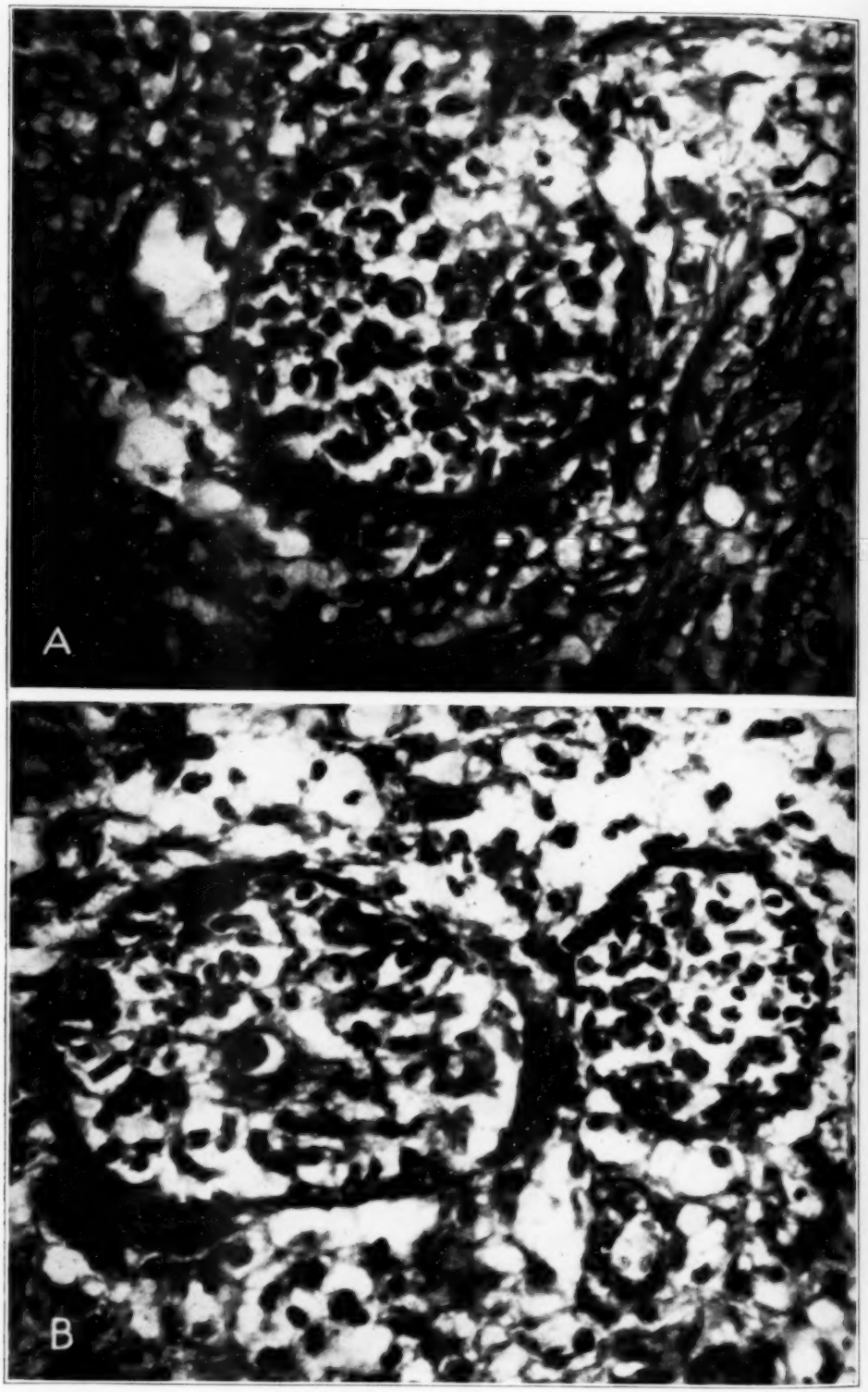


Fig. 2.—*A*, fibroblasts encapsulating an abscess; *B*, fibroblasts growing in between the leukocytes of an abscess.

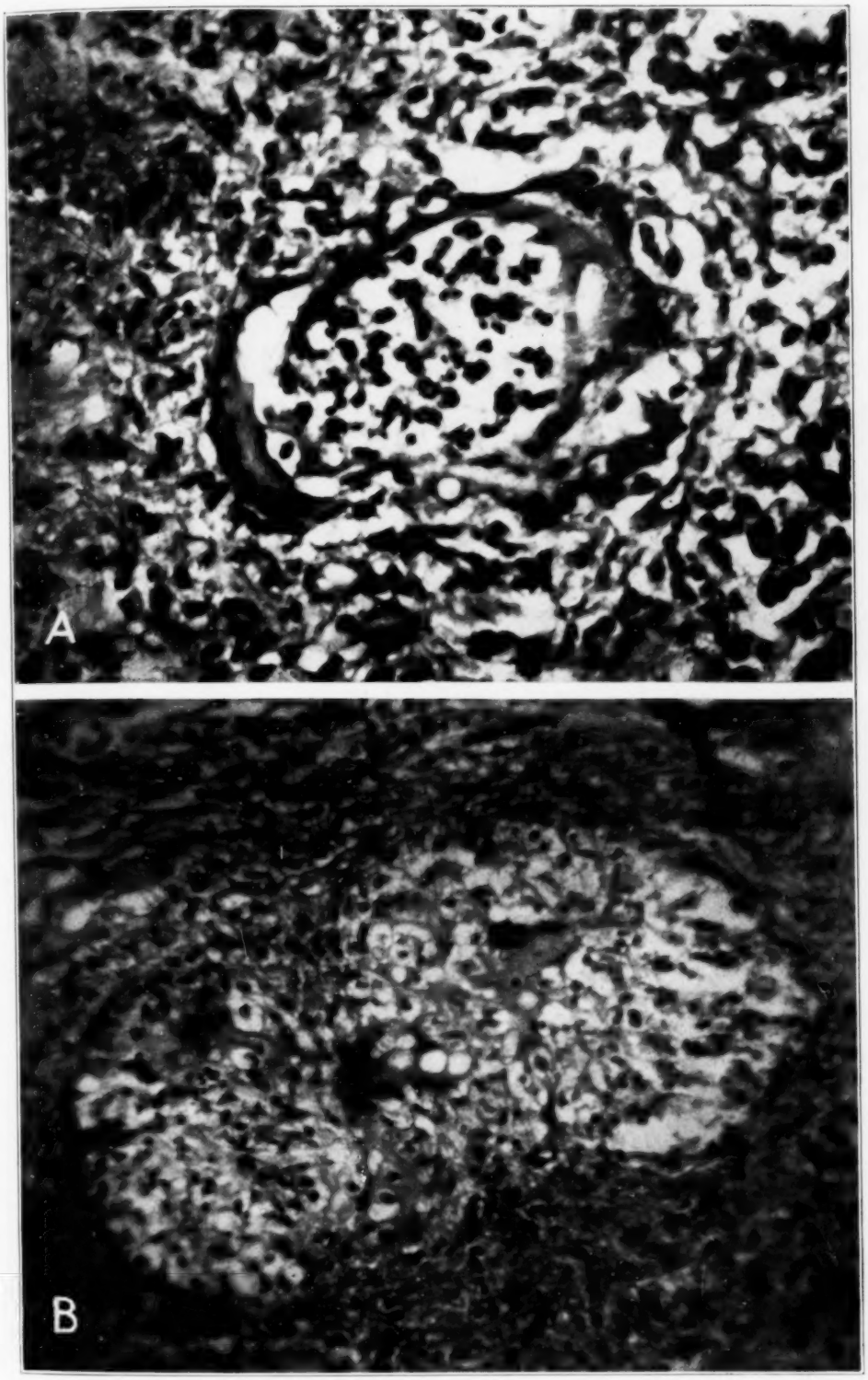


Fig. 3.—*A*, half-moon shaped giant cells surrounding an abscess; *B*, a focus in the tumor that resembles a tubercle somewhat. Note the leukocytes in the central area, the missing lymphocytic zone and the leukocytes in the surrounding tissue.

Areas were to be seen in the tumor which, on superficial inspection, somewhat resembled a tubercle (fig. 3 *B*). Careful examination, however, always showed numerous leukocytes in the central part, but never lymphocytes. The fibroblasts among the giant cells were usually arranged in circles, possessed fibers and formed a capsule of varying thickness around the abscesses.

To show the parasites, I prepared sections with hematoxylin-eosin (fig. 4 *A*), Giemsa's stain, Mallory's eosin-methylene blue and the Ziehl-Neelsen method. I also used Levaditi's method which I consider to be the best. Miller[4] had good results with Bielschowsky's silver impregnation method, but the pale yellow background of a Levaditi section brings the black organisms out with greater prominence. In sections prepared with Levaditi's method, the smaller parasites often appeared uniformly brownish-black, while the protoplasm of larger ones showed a fine or coarse brown granulation (fig. 4 *B*). Some spheres contained vacuoles. In the fresh abscesses, I found only a few round bodies; in the older foci, they were enclosed in larger numbers by the giant cells, and they were abundant on the edges of the necrotic areas. Budding organisms were also seen (fig. 5 *A*). Cultures were impossible, because the whole tumor had been preserved in solution of formaldehyde. The diagnosis, based on the morphologic examination of the fungus, is therefore not entirely conclusive.

COMMENT

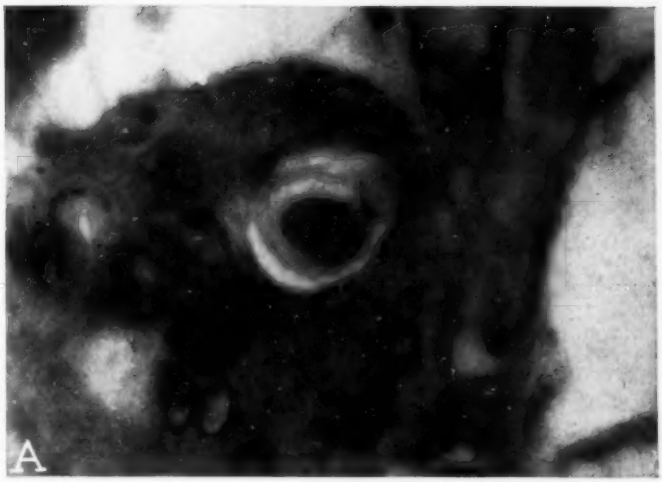
The tumor, according to the histologic examination, is a chronic blastomycotic meningitis with the formation of a large granulomatous mass that infiltrates the brain substance; in other words, the case is one of chronic blastomycotic meningo-encephalitis.

The earliest authors, Gilchrist and Stokes,[1] mentioned the formation of tubercle-like nodules, but did not discuss the differentiation from tuberculosis. Le Count and Meyers described the close resemblance of the blastomycotic lesions in the lungs to those of tuberculosis, but they also recorded the differences in the histologic structure between the two conditions. Hektoen[5] pointed out that the blastomyces may induce the formation of an inflammatory granulation tissue, in which either the suppurative or the proliferative processes may predominate. Histologically, the lesions, which show marked proliferative changes, "present certain general resemblances to those of tuberculosis. Real caseation and wholly typical tubercles, however, do not seem to occur." The older lesions may show some necrosis in the center and phagocytic giant cells are found in the walls of blastomycotic abscesses. Stober[6] stated that the central necrosis was not so marked in the blastomycotic nodules as in tuberculosis. The nodules were small abscesses, the larger ones often being surrounded by a distinct wall of granulation

4. Miller, W. S.: The Reticulum of the Lung: Its Similarity in Blastomycosis to that in Tuberculosis, Am. J. Path. **3**:315, 1927.

5. Hektoen, L.: Systemic Blastomycosis and Coccidioidal Granuloma, J. A. M. A. **49**:1071 (Sept. 28) 1907.

6. Stober, A. M.: Systemic Blastomycosis, Arch. Int. Med. **13**:557 (April) 1914.



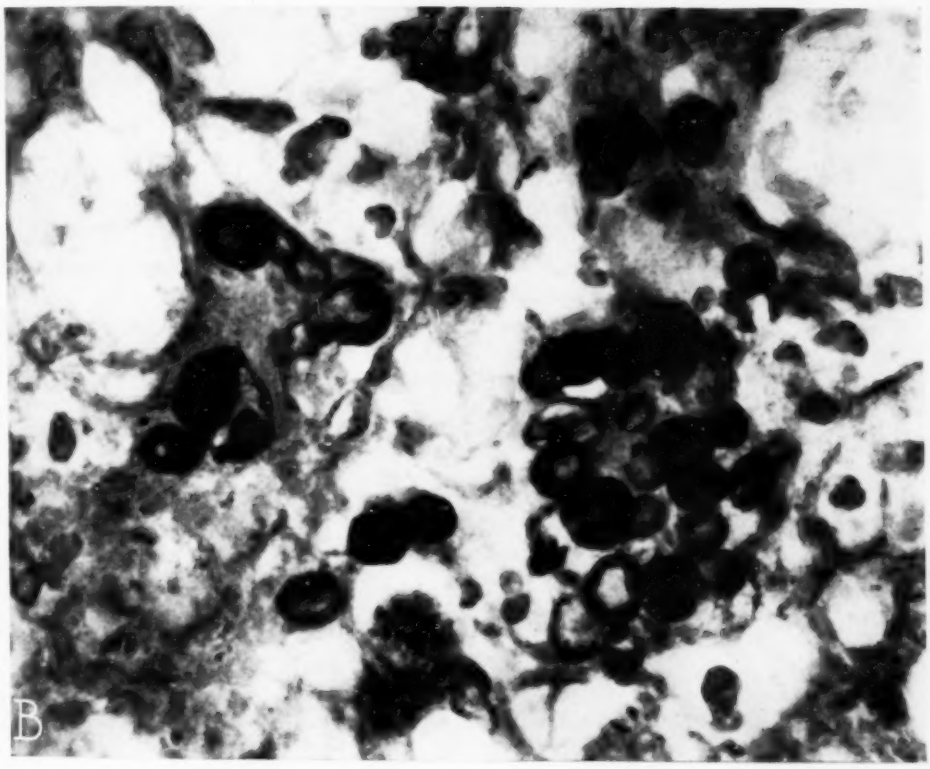


Fig. 4.—*A*, the parasite, apparently budding in a giant cell, as it looks with hematoxylin-eosin stain; *B*, the appearance of the blastomyces in section, prepared with Levaditi's method.

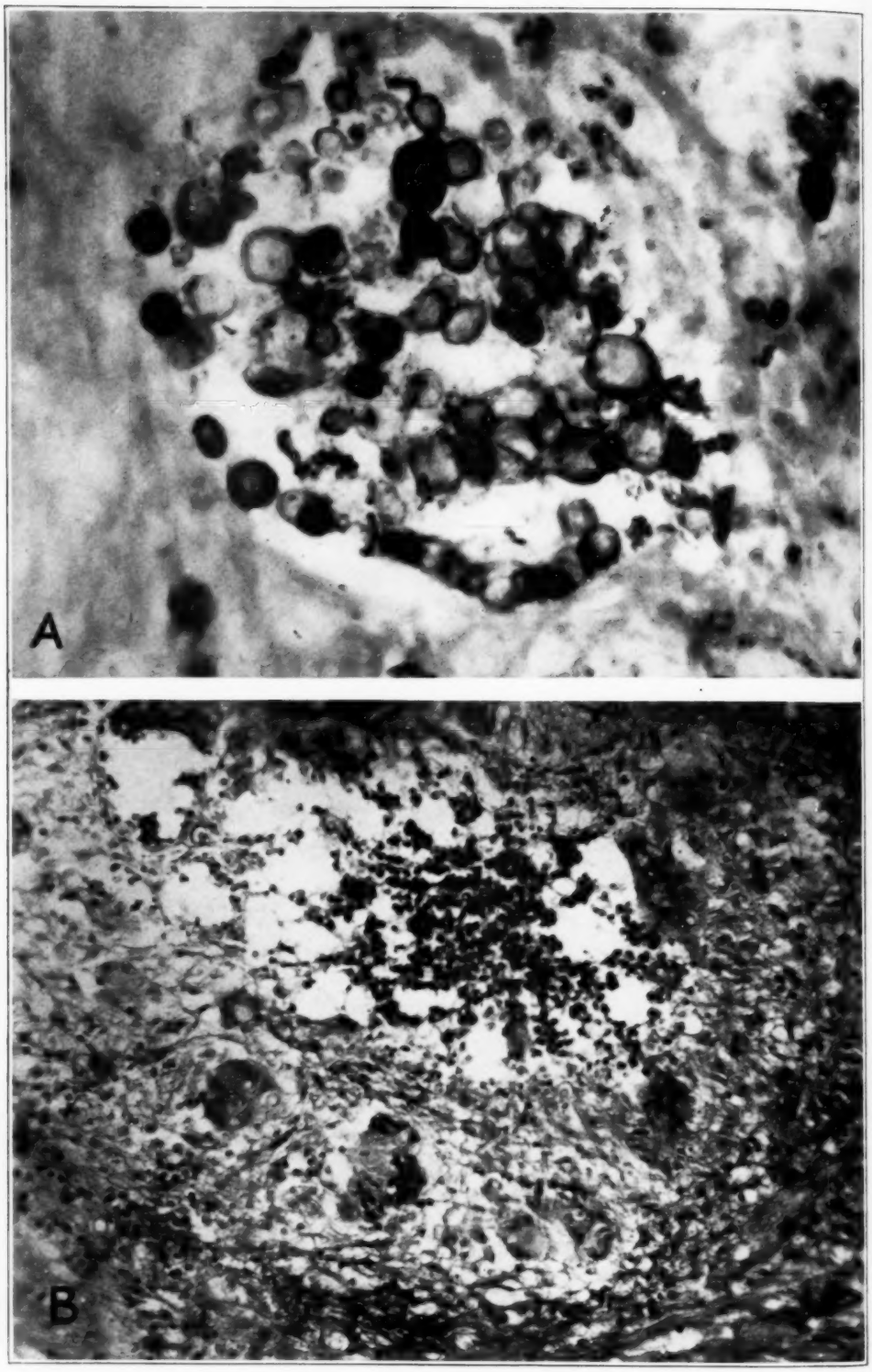


Fig. 5.—*A*, budding forms, Levaditi's method; *B*, larger abscess, around which there is an area very nearly identical with a tubercle, the outer indefinite zone being formed by lymphocytes, plasma cells and leukocytes.

tissue. The giant cells, if present, were rarely as typical as those found in tuberculosis. Healing occurs by marked fibrosis, especially in the skin, lungs and pleura.

Contrary to these investigators, Stoddard and Cutler noted that they found that the blastomycotic lesions so closely resemble tuberculosis that a differential diagnosis would be impossible without finding the parasites. Medlar,[7] in a recent paper, emphasized the identity of the gross and microscopic pathologic changes in blastomycotic and tuberculous lesions. He concluded, furthermore, that for a positive diagnosis of tuberculosis the demonstration of the tubercle bacilli or the absence of a fungus is necessary. "The presence of the parasite and not the pathologic lesion will establish the type of infection."

In my case I could follow microscopically in the same slides the whole infectious process in its various stages from the earliest to the oldest lesions and to necrosis. Thus, the whole range has been observed, and the slides have been compared with a large number of sections showing tuberculosis in various organs.

In my material the lesions produced by this organism were primarily abscesses; they may be miliary or larger. The abscesses slowly become encapsulated, and later, giant cells develop apparently among fibroblasts and engulf the parasites. This, in brief, is the sequence of formation of the different histologic lesions.

When the parasites appear in the uninfected tissue, leukocytes gather around them, and their numbers vary according to the number of the parasites. If only a few parasites are present the abscesses are small. When the parasites are numerous the abscesses are large. The abscesses represent therefore the acute stage of the disease.

The next step is the encapsulation of the abscesses. During this stage, the parasites are being engulfed by foreign body giant cells. This procedure shows numerous developmental grades and consists of a central abscess surrounded by a circular fibroblastic layer of varying thickness. When the giant cells appear they are to be found usually around the abscesses. Giant cells however, may appear near or in the center of the abscesses, and the focus may resemble that caused by the tubercle bacillus (fig. 5 *B*). This, however, is only a resemblance and is far from being identity, since here only leukocytes are to be found among the fibroblasts. The lymphocytic ring of tuberculosis is missing. Around larger abscesses, one may find a zone formed by many giant cells and fibroblasts, with an indistinct layer of plasma cells, lymphocytes and leukocytes in the outer inflammatory connective tissue. Even in such nodules, however, there is an abscess in the center. The outer zone

7. Medlar, E. M.: Pulmonary Blastomycosis: Its Similarity to Tuberculosis. Report of Two Cases, Am. J. Path. **3**:305, 1927.

is nearly identical with that found in a tubercle. The indistinct outer zone belongs rather to the inflammatory tissue than to the nodule, because one finds it only occasionally, especially when the process is more acute. The older nodules are not surrounded even with such an indefinite layer of round cells.

If the abscess is not large, it heals, and the giant cells are embedded in a hyaline fibrous tissue. Surrounding the giant cells, one still can find leukocytes, among which are eosinophil cells. Usually, when the parasites are numerous, necrosis takes place, involving giant cells, leukocytes and the fibroblasts. Epithelioid cells and lymphocytes are often entirely absent from the necrotic areas, the necrosis being enclosed only by a hyaline connective tissue.

On the basis of these histologic observations, I am unable to accept the opinion of several authors that the blastomycotic lesion, cannot be distinguished microscopically from that caused by the tubercle bacillus.

The macroscopic appearance of the tumor also shows differences from a tubercle. The rough surface on the side toward the brain, the small caseous spots on cut section, the boardlike toughness are not characteristic of a solitary tubercle, which is rather well outlined and uniformly caseous.

SUMMARY

A case of chronic blastomycotic meningo-encephalitis is presented.

Microscopically, the lesions are primarily abscesses, which slowly become encapsulated. The parasites are engulfed by the giant cells.

Histologically, the nodules may give the superficial impression of tubercles, but there is either an abscess or a varying number of leukocytes in the center. Lymphocytes rarely have been found. An outer zone, like that of a tubercle, exists only rarely in those areas which are close to an acutely inflamed focus, and even here numerous leukocytes and plasma cells are among the few lymphocytes.

The rampart of round cells seen around the focus in a tubercle is entirely missing in the large majority of the blastomycotic foci.

The lesions caused by the blastomyces can be distinguished histologically from the tubercle without demonstrating the characteristic organisms.

STUDIES IN STUTTERING

V. A STUDY OF SIMULTANEOUS ANTITROPIC MOVEMENTS OF THE HANDS OF STUTTERERS *

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AND
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IOWA CITY

A previous study [1] of this series showed that right-handed stutterers differed strikingly from right-handed normal speakers in the precedence of the action currents in the forearms during simultaneous voluntary flexion of the digits. These observations led us to make a study of the time of initiation of simultaneous antitropic movements of the hands in the two groups. We attempted to determine whether both hands start to move at exactly the same instant when the subject attempts to move them at the same time but in opposite directions. This apposition of direction was chosen in order to make use of the similarity of opposite movements which results from strict antitropism, that is, right and left pairing of the neuromuscular mechanisms.

TECHNIC

To record the movements of the two hands, we used a high speed kymograph. The drum revolved about a horizontal axis and carried a paper 8 feet (243.86 cm.) long and 1 foot (32.53 cm.) wide. Two heavy upright rods supported a pair of horizontal bars about 4 feet (121.92 cm.) from the floor. On one of these bars two easily sliding styli operated between collars equidistant from a central collar. The second horizontal bar served as a support for the styli. A 100 double vibration, electrically driven tuning fork furnished a time line (*T.*). To obtain the kymographic records, the subjects were instructed to grasp the styli and stand squarely in front of them. At the first command, "Go," they were to move the styli out (abduction), and at the second command they were to move the styli in (adduction). A line of reference, or base line, was made after each movement when the drum had been stopped. This base line was necessary for reading the records and was made by a complete motion out and back. Each subject was permitted to practice once before the drum was put in motion.

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* Submitted for publication, Feb. 1, 1929.

* The work here recorded was carried out in the laboratories of the Iowa State Psychopathic Hospital, under the direction of Dr. Samuel T. Orton, as a part of the program of research in the physiology of the brain. It was supported by a grant from the Rockefeller Foundation; additional funds were also supplied by the Graduate College of the State University of Iowa.

* This study was made while the senior author was a Fellow of the National Research Council.

1. Orton, S. T., and Travis, L. E.: Studies in Stuttering: IV. Studies of Action Currents in Stutterers, Arch. Neurol. & Psychiat. **21**:61 (Jan.) 1929.

The Parson manoptoscope[2] was employed to determine eyedness. A person is considered right-eyed or left-eyed according to the eye which he uses for sighting in binocular vision. We have termed a person amphiocular if he displays no fixed preference for either eye in sighting.

RECORDS

The kymographic records show the "start" or the initiation of the movement and the "arrival" or the conclusion of the movement. The "arrivals" did not show sufficient constancy with themselves or with the "starts" to be of value. Therefore we considered only the "starts."

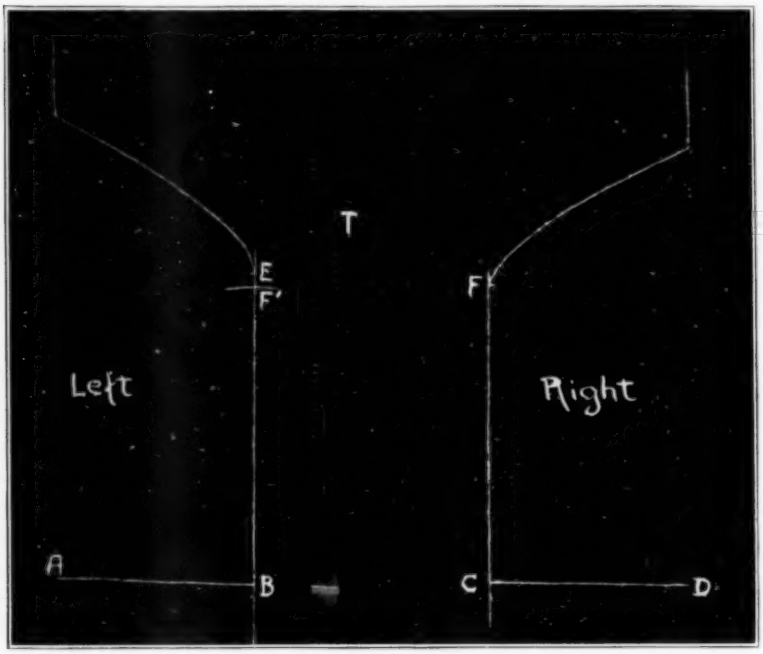

Fig. 1.—This and the following figures present kymographic records of abduction of the two hands in voluntary attempts to move both hands outward simultaneously. Time (*T*) is recorded in hundredths of a second. In this figure the movement of the right hand is recorded as preceding that of the left by about 0.015 seconds. This has been termed a right lead.

In the kymographic records, figures 1, 2 and 3, the horizontal lines *AB* and *CD* are base lines made by movement of the left and right hands, respectively, when the drum was not in motion. The vertical lines *BE* and *CF* are those made when the drum was in motion but the hands were at rest; by extending these lines it is possible to determine the instant of beginning movement from the point

2. Manoptoscope is the trade name of an instrument manufactured by C. H. Stoelting Company, Chicago, which takes advantage of the unilateral sighting in binocular vision. For a full description of this instrument, see Parson: Left-handedness, New York, The Macmillan Company, 1924.

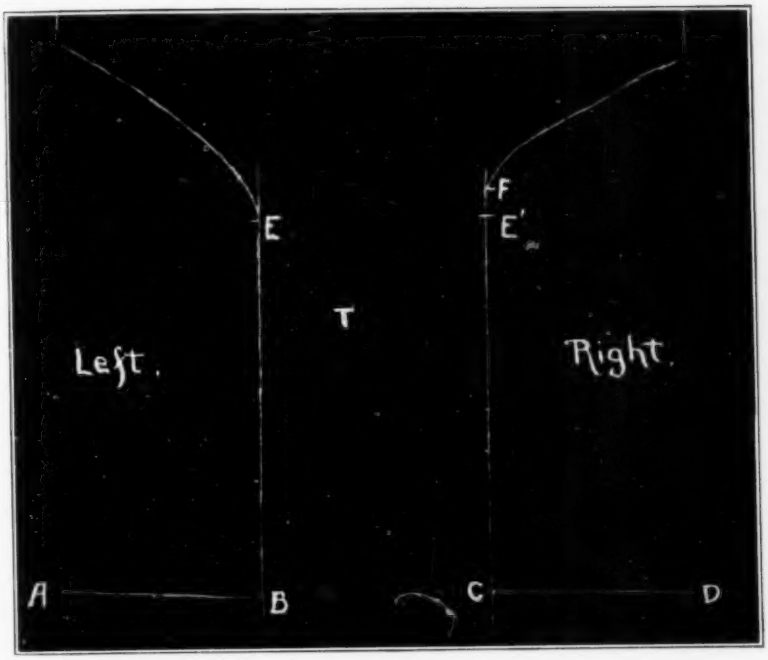

Fig. 2.—The movement of the left hand is recorded as preceding that of the right hand by about 0.02 second. This has been termed a left lead.

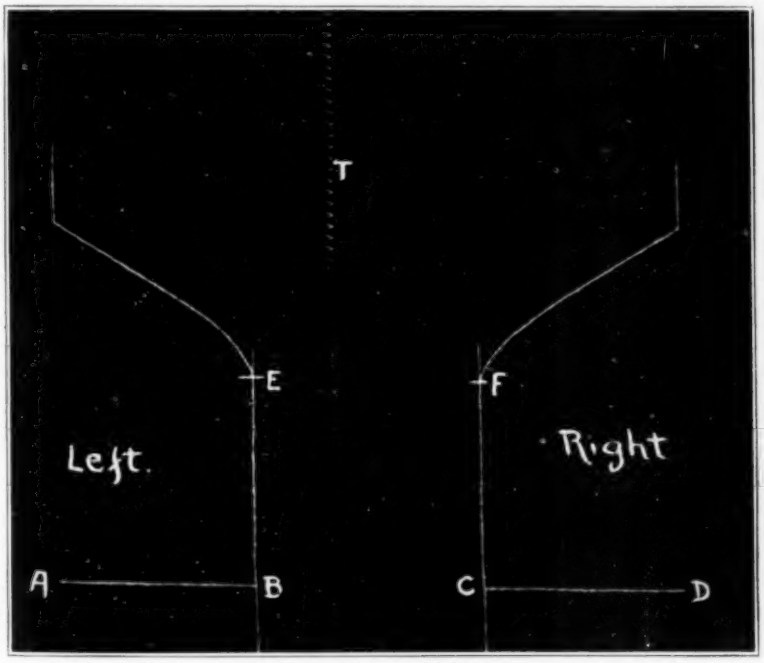

Fig. 3.—The movements of the two hands are recorded as appearing simultaneously. This has been termed a simultaneous lead.

where the curve of motion leaves these lines of rest. These points are marked E for the left hand and F for the right. By projecting the shorter distances from the base line on the longer, one can determine, with considerable accuracy, by comparison with the time line in the center the difference in the time of initiation of motion in the two hands. Thus, in figure 1, if CF is projected on BE one obtains the point F'; the distance $F'E$, which equals approximately 0.015 seconds, represents the time which elapsed after the right hand movement began and before the left hand got under way. This we term a right lead. In figure 2, the distance CF is the longer and if we project BE on CF we get the point E'; the distance $E'F$, about 0.02 seconds, constitutes a left lead. In figure 3, the two hands got under way at exactly the same instant. This we term a simultaneous lead.

All of the figures present abduction movements. The method for determining the lead in adduction movements is the same as that just described. In the tables adbduction and adduction movements are considered together.

TABLE 1.—*Right-Handed Normal Speakers with a History of Right-Handedness*

Person Tested	Total Number of Trials	Right Lead	Left Lead	Simultaneous Lead	Eyedness
1	45	6	33	6	Left
2	30	6	24	0	Right
3	11	0	11	0	Right
4	17	1	15	1	Right
5	10	1	7	2	Right
6	5	0	5	0	Right
7	10	0	10	0	Right
8	22	2	16	4	Right
9	19	2	13	4	Right
10	18	0	15	3	Right
11	20	1	19	0	Left
12	20	3	13	4	Right
13	18	2	16	0	Right
14	26	16	10	6	Left
15	19	15	3	1	Right
16	19	12	5	2	Right
17	22	12	8	2	Left
18	20	6	13	1	Right
Totals	351	79	236	36	
Per cent of total		22	68	8	

MATERIAL STUDIED AND RESULTS

None of the persons listed as normal speakers had any observable defect of speech of any type. The stutterers presented defects varying from a mild degree to a severe degree of stuttering. With the exception of four stutterers (nos. 5, 6, 11 and 13, table 5), who were children in elementary schools, all subjects were adults less than 30 years of age. Nine normal speakers (8, 10, 12, 13 and 14, table 1; 12, 13 and 14, table 2; and 9, table 3) and one stutterer (17, table 4) were females.

The normal speakers entered in table 1 showed a preferential choice of the right hand in carrying out all complex motor functions and gave a history of having always indicated a preference for the right hand. The table presents a marked predominance of left leads and of right-eyedness. In only three persons (15, 16 and 17) did the number of right leads exceed the number of left leads. It would seem then that

normal speakers who have always met the ordinary criteria of right-handedness lead more frequently with the left hand when both hands are voluntarily moved out or in simultaneously, and they are more apt to be right-eyed.

The normal speakers listed in table 2 showed at the time of the experiment a preferential choice of the right hand in all motor functions, but gave histories of having been left-handed in childhood, accord-

TABLE 2.—*Right-Handed Normal Speakers with a History of Left-Handedness*

Person Tested	Total Number of Trials	Right Lead	Left Lead	Simultaneous Lead	Eyedness
1	5	2	3	0	Right
2	12	7	4	1	Right
3	8	5	2	1	Right
4	7	2	5	0	Left
5	20	9	9	2	Right
6	9	3	4	2	Right
7	14	8	5	1	Right
8	13	8	4	1	Left
9	19	13	5	1	Left
10	9	6	3	0	Right
11	20	8	9	3	Left
12	12	2	8	2	Amphiocular
13	14	0	13	1	Right
14	13	3	7	3	Left
Totals	175	76	81	18	
Per cent of total..		43	46	11	

TABLE 3.—*Left-Handed Normal Speakers with a History of Left-Handedness*

Person Tested	Total Number of Trials	Right Lead	Left Lead	Simultaneous Lead	Eyedness
1	20	16	4	0	Left
2	12	8	0	4	Left
3	6	5	0	1	Left
4	9	8	1	0	Right
5	7	6	1	0	Left
6	14	11	2	1	Left
7	18	13	4	1	Left
8	10	6	3	1	Right
9	25	7	16	2	Right
10	9	5	4	0	Left
11	15	11	4	0	Right
12	14	5	9	0	Left
Totals	159	101	48	10	
Per cent of total..		64	30	6	

ing to the ordinary criteria. They furnished no significant difference in the number of right and left leads, although each of these leads was significantly greater in number than simultaneous leads. Also, there was not the marked preponderance of right-eyedness in this group that was evidenced in the other group.

The normal speakers listed in table 3 fulfilled the common criteria of having always been left-handed. This group presented a significantly greater number of right than of left or simultaneous leads and a majority were left-eyed. Only two gave a greater number of left than of right

leads. Thus, it appears that the purely left-handed normal speaker leads more frequently with the right hand and is more apt to be left-eyed, as contrasted with the purely right-handed normal speaker who leads more often with the left hand and is more likely to be right-eyed.

TABLE 4.—*Right-Handed Stutterers with a History of Right-Handedness*

Person Tested	Total Number of Trials	Right Lead	Left Lead	Simultaneous Lead	Eyedness
1	18	14	3	1	Amphiocular
2	24	22	2	0	Right
3	50	45	4	1	Right
4	16	12	4	0	Right
5	40	27	9	4	Right
6	26	19	6	1	Right
7	14	7	7	0	Amphiocular
8	17	7	10	0	Left
9	22	5	14	3	Right
10	30	18	11	1	Left
11	32	18	14	0	Amphiocular
12	35	8	22	5	Left
13	26	12	11	3	Right
14	31	7	20	4	Amphiocular
15	21	10	10	1	Right
16	9	7	1	1	Right
17	9	7	1	1	Amphiocular
18	6	0	4	2	Left
Totals	426	245	153	28	
Per cent of total..		57	35	8	

TABLE 5.—*Right-Handed Stutterers with a History of Left-Handedness*

Person Tested	Total Number of Trials	Right Lead	Left Lead	Simultaneous Lead	Eyedness
1	27	5	22	0	Left
2	19	13	4	2	Left
3	11	4	4	3	Right
4	19	10	7	2	Left
5	69	27	33	9	Right
6	34	20	12	2	Amphiocular
7	18	10	7	1	Right
8	19	10	9	0	Amphiocular
9	27	12	11	4	Right
10	9	5	0	4	Left
11	14	9	2	3	Right
12	25	17	7	1	Right
13	24	21	2	1	Right
Totals	315	163	120	32	
Per cent of total..		52	38	10	

All of the stutterers (tables 4, 5 and 6), although they were to be rated as right-handed by the usual tests, including writing, gave results from the movements similar to those of the purely left-handed normal speakers and results from the tests of eyedness comparable to those of the right-handed normal speakers with a history of left-handedness. Tables 4, 5 and 6 indicate that neither a positive nor a negative history of left-handedness in stutterers means much. It indicates certainly that a history of right-handedness means nothing.

A comparison of the groups is shown in table 7.

This study bears out the one on action current in that right-handed stutterers differ materially from purely right-handed normal speakers in regard to motor leads as determined by these two studies. It goes further in showing that right-handed stutterers are similar to purely left-handed normal speakers when compared on the basis of the determined

TABLE 6.—*Right-Handed Stutterers on Whom No History of Handedness Was Obtainable*

Person Tested	Total Number of Trials	Right Lead	Left Lead	Simultaneous Lead	Eyedness
1.....	21	16	4	1	Left
2.....	37	19	13	5	Right
3.....	24	14	5	5	Left
4.....	7	4	2	1	Amphiocular
5.....	15	5	10	0	Right
6.....	36	17	15	4	Right
7.....	20	11	6	3	Right
Totals	160	86	55	19	
Per cent of total..		54	31	15	

TABLE 7.—*Summary of Tables*

Groups Tested	Number	Right Lead,	Left Lead,	Simultaneous Lead,	Eyedness		
					Right,	Left,	Amphiocular,
Righthanded normal speakers with a history of right-handedness	18	22	68	8	78	22	..
Righthanded normal speakers with a history of left-handedness	14	43	46	11	57	36	7
Lefthanded normal speakers with a history of left-handedness	12	64	30	6	33	67	..
Righthanded stutterers with a history of righthandedness	18	57	35	8	50	22	28
Righthanded stutterers with a history of lefthandedness	13	52	38	10	54	31	15
Righthanded stutterers on whom no history was obtainable	7	54	31	15	60	30	10

motor lead. Again, it bears out our former inference that in many stutterers the motor facility, as determined by training, is out of harmony with the native physiologic leads. Why purely right-handed normal speakers lead with the left hand and purely left-handed normal speakers lead with the right in these abduction and adduction movements is beyond the scope of this study. That they do and that the right-handed stutterers respond similarly to the purely left-handed normal subjects is the significant fact for the present.

SUMMARY

In simultaneous antitropic movements of the two hands (abduction and adduction), we found the following to be true of the groups studied: Right-handed normal speakers with a history of right-handedness lead more frequently with the left hand; right-handed normal speakers with a history of left-handedness lead about as often with one hand as with the other; left-handed normal speakers with a history of left-handedness lead more frequently with the right hand, and right-handed stutterers, regardless of a positive or a negative history of left-handedness, lead more frequently with the right hand. No significant differences seemed to be found between the groups, relative to simultaneous leads.

In a supplementary study of eyedness, we found a significantly greater number of amphiocular persons among the right-handed stutterers than among any other group, with the possible exception of the right-handed normal speakers with a history of left-handedness. There were fewer right-eyed subjects among the right-handed stutterers than among the right-handed normal subjects with a history of right-handedness.

THE QUANTITATIVE DISTRIBUTION OF THE PYRAMIDAL TRACT IN MAN*

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It is an axiom, generally recognized in neurophysiology, that the development of skilled acts in the upper and lower extremities of the highest forms of animal life is intimately connected with the size of the pyramidal tracts. The work of Schaefer [1] is quoted as giving experimental evidence of this thesis, and Tilney [2] concluded from comparative studies of the central nervous system of apes and man that "it is in relation to the progressive adaptation apparent in the upper extremities that the pyramidal system is most intimately connected. . . . It seems permissible to conclude that the increment in pyramidal volume is proportional to that increasing demand for control of motor performances made possible through the progressive development of the hand."

In studying the literature on this subject one finds that comparative measurements of the pyramids in the medulla oblongata and the size of the nonmedullated tract in new-born animals form the anatomic basis for these theories. The planimetric index, i.e., the size of the tract in transverse sections as compared in percentages with the total plane of the section expressed in square centimeters, furnishes a concrete medium of comparison. One of us (A. W.) has shown recently [3] that in sections through the medulla oblongata at the caudal end of the inferior olives the planimetric index is from 3 to 4.8 for marsupials, ungulates and rodents; from 7.7 to 8.3 for carnivora (cat) and lower primates (*Tarsius*) and from 10.2 to 11.3 for the gibbon, the higher primates and man.

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1. Schaefer, E. A.: Experiments on the Paths Taken by Volitional Impulses Passing from the Cerebral Cortex to the Cord: The Pyramids and the Ventro-Lateral Descending Tracts, Quart. J. Exper. Physiol. **3**:355, 1910.

2. Tilney, F.: The Brain from Ape to Man, New York, Paul B. Hoeber, 1928.

3. Weil, A.: A Comparative Quantitative Study of Proprioceptive Tracts of Mammals, J. Comp. Neurol. **44**:61, 1927.

The planimetric index, however, is only a relative value which indicates the relation of a given tract to the total number of nerve fibers at a given plane, but which does not allow any conclusions as to its absolute size and distribution. No investigations seem to have been made to determine the relationship between the size and the weight of the musculature which is supplied by the pyramidal tract and the size and the number of fibers of the tract itself. Such data would give an anatomic basis for control of statements on the importance of the pyramidal tract drawn from physiologic observation. At the same time such data would be of interest to the neurologist in the explanation of phenomena noted in patients with lesions of the pyramidal tract.

THE PROBLEM

Our original problem was to determine the size of the pyramidal tract in man at each segment of the spinal cord and the amount of striated musculature supplied from nerve fibers originating at these segments. New problems arose while the work was in progress: the degree

TABLE 1.—*Analysis of Thirty-Eight Cases of Hemiplegia and Diplegia*

Disease	Number of Cases	Percentage	Right	Left	Right and Left
Vascular disease	29	76	12	11	6
Tumors					
(a) Primary	2	5	..	1	1
(b) Metastatic	7	19	4	2	1
Total	38	100	16	14	8

of condensation of pyramidal tract fibers at different levels of the spinal cord; the amount of shrinkage encountered in the different methods of embedding and staining; the relationship between the size of the field of demyelinization and the field of glial reaction, and the weight and distribution of striated muscles in persons of different age and sex.

THE METHOD

The material was obtained from the neurologic service of the Montefiore Hospital, New York, through Dr. W. M. Kraus and Dr. C. H. Davison and included cases which clinically had shown hemiplegia or diplegia. The analysis of these thirty-eight cases is given in table 1.

Ten cases were finally selected which on anatomic examination showed localized lesions in the region of the internal capsule, or destruction of the precentral area. Case 1 is an exception and presents a well localized, organized softening in the right anterior part of the pons, comprising the bundles of the pyramidal tract. Case 8 is one of hemorrhage into the left side of the pons and cerebellum. All the lesions were vascular in origin (arteriosclerosis) and had a duration of from one and one-half to eleven years, with the exception of cases 6 (six months) and 8 (four months). Sufficient time, therefore, had elapsed for the development of complete secondary degeneration of the pyramidal tract and for the formation of a final glial scar.

Care was taken to obtain the spinal cords as completely as possible, though in many cases a loss of part of the higher cervical segments, due to the technic of sectioning, could not be avoided. The various segments of each cord were embedded in the same way; i. e., they were left in the same amount of alcohol the same amount of time and finally were embedded in one large dish of 6 per cent collodion preparation solution in order to obtain an even shrinkage of the embedding material. Sections of 20 microns were cut and stained with the Loyez method, or its rapid modification,[4] and with Mallory's or van Gieson's method. The degenerated area, in sections stained to show the myelin sheaths, is not always sharply demarcated, and only one third of the material could be utilized for this reason. In the remainder of the cases either the outlines of the degenerated pyramidal tract were indistinct, or numerous myelinated fibers were intermingled. In the ten cases finally selected, different measurements by different examiners gave maximal errors of about 10 per cent. The degenerated area was copied on glazed paper on the ground glass of a photomicrograph apparatus, which enlarged it about thirty-five times, and this area was measured either with the help of a planimeter,

TABLE 2.—*Ten Cases Selected with Localized Lesions in the Internal Capsule or Destruction in the Precentral Area*

No.	Age	Sex	Duration of Hemiplegia	Hemiplegia	Anatomic Lesion
1	57	F	1½ years	Left	Pontile softening
2	50	M	8 years	Left	Cyst in right internal capsule
3	71	M	11 years	Left	Organized hemorrhage in right internal capsule
4	74	M	2 years	Right	Organized hemorrhage in left internal capsule
5	63	M	2½ years	Left	Organized softening in region of right middle cerebral artery
6	65	F	6 months	Left	Hemorrhage into right internal capsule
7	67	F	2½ years	Left	Organized softening in region of right middle cerebral artery
8	64	M	4 months	Left	Hemorrhage into pons and cerebellum
9	60	M	4 years	Left	Anemic softening in region of right striatum and internal capsule
10	56	F	2 years	Right	Anemic softening of left striatum and internal capsule

or by counting the squares over millimeter paper. The absolute value of the area was found by dividing the square millimeters by the square of the linear enlargement.

For counting and measuring the nerve fibers, blocks were either stained by Bielschowsky's method (pyridine modification) and then embedded in paraffin, or sections, 10 microns thick, of paraffin-embedded formaldehyde material were stained by the rapid modification of Loyez' method. In blocks treated by the Bielschowsky method there is a considerable degree of shrinkage as compared with the formaldehyde material. The shrinkage is most pronounced in the cervical and lumbar segments, due perhaps to a larger proportion of gray matter, and therefore of protein which is precipitated by the silver nitrate. For comparative purposes all measurements in Bielschowsky-stained sections were recalculated for shrinkage, as compared with the corresponding sections from paraffin material stained for myelin sheaths. The degree of shrinkage was determined by measuring the anterior-posterior diameter of a section at high magnification. Photographs were taken with an oil immersion objective and a Leitz "periplan" ocular (× 8) on contrast plates and a contrast developer was used. Contact copies were made on lantern slides and projected with a final linear enlargement of 10,000

4. Weil, A.: A Rapid Method for Staining Myelin Sheaths, Arch. Neurol. & Psychiat. **20**:392 (Aug.) 1928.

times. For the counting of axons or myelinated fibers the negatives were placed on top of a light-box and covered with thin glazed paper, on which each fiber was marked with a pencil. The area measured was always the same, a circle 122 mm. in diameter, corresponding to the mask which was used in the photomicrograph apparatus. The number of fibers counted was multiplied by the plane of this circle measured in square millimeters, and the product was divided by the square of the linear enlargement.

The number of a segment indicates the rostral part of that segment, with the exception of the first cervical segment, in which sections of the more distal parts were stained.

To determine the weight of human muscles, the average weights of nine men and six women were calculated from Theile's tables.[5] Judging from results of the relative distribution of male and female muscles (table 6), Theile's measurements indicate a high degree of accuracy. For the determination of segmental innervation of the muscles we followed the tables given by Spiegel.[6] First preference was given to Foerster's data, or if such were not available for a certain muscle, Bing's [7] or Cunningham's [8] data were used. When several segments were given as the supply of a muscle, the weight of that muscle was divided by the number of segments, and a proportionate weight was allotted to each segment. As a basis for the construction of figure 3 this method appears to be more or less arbitrary; however, the final results of table 6, in which the muscles were arranged in large groups, were not influenced by this subdivision.

RESULTS

The figures of table 3 were obtained by measuring the area of degeneration in sections stained by Loyez' method. Comparative studies with sections stained with Weigert's (Pal-Kulschitzky) method and with Loyez' method showed that there was no appreciable difference in the size of the demyelinated areas, or in the number of myelinated fibers counted in normal tracts, if compared with sections from the same level stained with the rapid modification of Loyez' method. The latter was given preference, not only on account of the shorter time, but also on account of the better preservation of the embedded material, which is not as fragile as the material which has remained for a week in Weigert's mordant.

In case 1, no first or second cervical segment was available. The value for the first cervical was calculated from the fourth cervical segment, assuming 80 per cent of the pyramidal tract at this level, which is the average of the other cases. The empty spaces in table 3 indicate that

5. Theile, F. W.: Gewichtsbestimmungen zur Entwicklung des Muskelsystems und des Skelettes beim Menschen, Verhandl. d. Kaiserl. Leopoldinisch Carolinischen deutsch. Akad. d. Naturforscher., 1884, vol. 46, part 3.

6. Spiegel, E. A.: Experimentelle Neurologie, Berlin, S. Karger, 1928, part 1, p. 185.

7. Bing, R.: Kompendium der topischen Gehirn und Rueckenmarksdiagnostik, ed. 6, 1925, Berlin, Urban & Schwarzenberg, 1925, p. 52.

8. Cunningham: Textbook of Anatomy, ed. 4, New York, William Wood & Company, 1915, p. 744.

TABLE 3.—*Area of Degenerated Pyramidal Tract in Square Millimeters*

	Segment	Case 1	2	3	4	5	6	7	8	9	10
Cervical	I	5.76	3.82	2.74	3.56	2.38	4.14	4.60	4.17	3.34	5.78
	II	....	....	....	....	2.29	....	....	3.87	....	....
	III	....	....	....	....	....	....	....	....	....	....
	IV	4.62	3.39	....	....	....	....	....	3.51	2.52	4.39
	V	4.10	....	....	....	1.88	3.45	3.22	....	....	3.91
	VI	....	2.73	1.80	2.39	....	....	3.05	....	1.98	....
	VII	....	....	....	2.18	1.39	....	....	2.89	1.88	....
	VIII	....	2.20	1.66	2.13	1.33	....	....	2.76	....	3.48
Dorsal	I	3.45	2.07	1.60	....	....	....	....	2.33	....	....
	II	3.18	....	1.30	....	1.24	2.00	2.50	2.06	1.54	....
	III	....	....	....	....	1.21	1.80	2.25	....	....	....
	IV	....	....	1.19	1.57	....	....	1.92	....	....	....
	V	2.92	1.69	1.12	1.36	....	....	1.81	....	1.36	2.86
	VI	....	....	1.00	1.32	1.03	....	....	....	....	....
	VII	2.66	....	0.98	....	....	....	....	....	....	....
	VIII	....	....	....	....	....	....	....	....	....	2.81
	IX	....	....	....	....	0.93	1.58	....	1.27	....	....
	X	1.90	....	....	....	0.84	....	....	....	....	2.54
	XI	....	1.28	0.78	1.22	0.80	....	....	1.25	....	....
	XII	1.74	....	0.70	1.19	0.76	....	1.36	....	....	2.07
Lumbar	I	....	1.14	0.78	1.16	0.75	1.24	1.33	1.17	0.91	....
	II	....	1.23	0.69	....	....	0.96	1.26	1.09	....	....
	III	....	0.97	0.54	0.87	....	....	1.18	1.00	....	....
	IV	0.90	....	....	0.56	0.60	....	....	....	....	....
	V	....	0.76	....	0.48	0.42	....	....	0.86	....	....
Sacral	I	....	....	....	....	....	0.26	0.62	....	....	....

TABLE 4.—*Percentage Values of Degenerated Pyramidal Tract Areas Giving the First Cervical Segment a Value of 100*

	Segment	Case 1	2	3	4	5	6	7	8	9	10	Average
Cervical	I	100	100	100	100	100	100	100	100	100	100	100
	II	...	...	...	...	96	...	...	93	...	...	95
	III	...	...	...	...	...	...	...	...	...	...	...
	IV	80	...	...	89	...	...	...	84	76	77	81
	V	71	...	...	...	79	83	72	...	...	68	75
	VI	...	71	66	67	...	...	66	...	...	...	67
	VII	...	...	...	61	58	...	...	69	56	...	61
	VIII	...	58	61	60	57	...	...	66	...	60	60
Dorsal	I	60	54	58	...	...	...	...	56	...	...	57
	II	55	...	47	...	52	48	54	49	46	...	50
	III	...	...	...	...	51	43	49	...	...	...	48
	IV	...	...	43	44	...	...	42	...	...	...	43
	V	51	44	41	38	...	...	39	...	41	50	43
	VI	...	...	37	37	43	...	...	...	...	...	39
	VII	46	...	36	...	...	...	...	...	...	...	41
	VIII	...	...	...	...	...	...	...	...	...	49	...
	IX	...	...	...	...	39	38	...	31	...	...	36
	X	33	...	...	...	35	...	...	...	...	...	34
	XI	...	33	28	34	34	...	...	30	...	...	32
	XII	30	...	26	33	32	...	30	...	...	36	31
Lumbar	I	...	30	28	32	31	30	29	28	27	...	30
	II	...	32	25	...	...	23	27	26	...	...	27
	III	...	25	20	24	...	...	26	24	...	...	24
	IV	16	...	...	16	25	...	...	...	...	...	19
	V	...	20	...	13	20	...	...	23	...	...	18
Sacral	I	...	...	...	...	...	6	13	...	...	...	10

either no material from these segments could be obtained, or that the degeneration was not clearly enough defined to allow an exact measurement, or that the segment itself had been distorted when it was removed.

Averages from ten different cases with different anatomic lesions will naturally give only a general idea of the percentage distribution of the pyramidal tract. Comparison of the averages with the two cases, 5 and 7, however, will show that the averages approach the values of these two cases closely, despite the large difference in absolute size of the degenerated areas.

The values for the size of the degenerated area of the anterior pyramidal tract are given in table 5. Only in four cases of hemiplegia could a definite area of degeneration be made out in myelin-stained sections. In four other cases there were only scattered spots of demyelinization in the area where one expects the anterior tract, but no well defined tract could be made out (cases 6, 7, 8 and 10). In two cases there was

TABLE 5.—*Relation of Crossed to Uncrossed Pyramidal Tract Fibers; Four Cases of Hemiplegia*

	Case 2	Case 3	Case 4	Case 5
Segment	Cervical IV	Cervical I	Cervical I	Cervical II
Plane in square millimeters				
(a) Lateral	3.82	2.74	3.56	2.29
(b) Anterior	0.60	0.32	0.30	0.19
Planimetric index				
(a) Lateral	12.1	10.4	10.8	9.3
(b) Anterior	1.9	1.2	0.9	0.8
Anterior tract in percentages of lateral tract	15.7	11.7	8.4	8.3

no evidence of degenerated pyramidal tract fibers in the anterior columns (cases 1 and 6).

The muscles were divided into six different groups according to their innervation by the various plexuses and thoracic nerves. The absolute values show a difference between male and female musculature of 31 per cent. The percentage distribution indicates the same distribution for the muscles of the head and neck in males and females, also for the abdominal muscles and the muscles supplying the leg. The male musculature, however, shows a relatively larger weight for the muscles of the shoulder and arm, while in the female there is a higher percentage for the muscles of the pelvis and thigh. Columns 6 and 7 (table 6) show that the part of the pyramidal tract fibers innervating the unit of muscle weight is the same in the male and female.

The numbers of table 7 are averages of several counts. In order to obtain comparable results it is necessary always to use photographs of the same area of the pyramidal tract. From in outward the number of nerve fibers per square millimeter decreases considerably, especially near the borderline of the spinocerebellar tracts. The photographs, therefore, were always taken at the border line of the inner and middle

third of the pyramidal tract, the position of which was determined in case 10 by comparison with the degenerated area of the other side. In the normal case (N), the position of the pyramidal tract fibers was determined by comparison with case 10. On close examination, such photographs will show the characteristic arrangement of the pyramidal tract fibers which permits their differentiation from the fiber tract of the periphery.

In evaluating the results of table 8 one should bear in mind that considerable shrinkage takes place during embedding and staining. Further-

TABLE 6.—*Distribution of Muscles in Relation to Segmental Innervation*

	Weight of Muscles of One Side in Grams		Percentage Distribution of Muscles		Percentage of Pyramidal Tract Fibers	Percentage of Pyramidal Tract Fibers Innervating 1 per Cent of Muscle*	
	Male	Female	Male	Female		Male	Female
Segments	Column 1	2	3	4	5	6	7
C I - IV	794	575	6.5	6.9	25	3.8	3.6
C V - D II	3,441	2,185	28.3	26.2	27	1.0	1.0
D III - D XI	825	618	6.8	7.4	17	2.5	2.3
D XII - L IV	3,526	2,270	29.0	27.2	13	0.45	0.48
L V - S IV	3,577	2,690	29.4	32.3	18	0.61	0.56
Total	12,163	8,338					

* The last two columns were calculated by dividing the numbers in column 5 by those in column 3 or 4, respectively.

TABLE 7.—*Nerve Fibers of the Pyramidal Tract; Number per Square Millimeter*

		Bielschowsky-Stained Fibers				
Case	Segment	Counted	Shrinkage, per Cent	Actual Number	Myelinated	Percentage Difference
10	C V	76,500	11	68,000	51,000	25
N	C V	73,000	8	67,000	.....	..
10	C VIII	69,500	8	64,000	51,000	20
N	D II	80,500	23	62,000	.....	..
10	D V	65,000	..	65,000	43,000	34
N	D IX	62,500	4	60,000	.....	..
10	D X	69,000	6	65,000	45,000	31
10	D XII	75,000	13	65,000	50,000	23
N	L I	77,000	14	66,000	.....	..

more, the number of small fibers which may be detected will always depend on the optical instruments and the technic used.

COMMENT

From a comparison of tables 2 and 4 it becomes evident that the size of the pyramidal tract lesion—either cortical or capsular—is without influence on the percentage distribution of the pyramidal tract fibers in the spinal cord. The size of the degenerated area is dependent on the age of the glial scar. The case of eleven years' duration (case 3) is one of the smallest, the area of degeneration being 2.74 sq. mm. at the first cervical segment. In case 5, of two and one-half years' duration, the

same area is 2.38 sq. mm. Case 10 cannot be compared with the rest as to absolute size, because the material was embedded in paraffin, and that of the other cases in a collodion preparation. The large size of the degenerated area in case 1 (pontile softening) throughout the spinal cord corresponds to observations in other cases of pontile or medullary pyramidal tract lesions. Ziehen [9] commented on this and pointed out that the higher the lesion within the brain, the smaller the area of degeneration. In case 1, the absence of an anterior tract is perplexing, but it may explain the large size of the lateral tract. One has to assume as a working basis that the degeneration and glial scar formation proceeds at an even speed throughout the whole spinal cord. That this assumption is justified follows from the close resemblance of the percentage distribution of the degenerated area in cases of different etiology and duration.

TABLE 8.—*Diameter of Nerve Fibers in Pyramidal Tract and Their Percentage Distribution*

	Bielschowsky Stained Segment		Myelinated Fibers Segment	
Size of Fiber, Microns	Cervical VIII, per Cent	Lumbar I, per Cent	Cervical VIII, per Cent	Lumbar I, per Cent
0.3 - 1	48	34	3	1
1.1 - 2	39	51	54	43
2.1 - 3	9	13	17	18
3.1 - 4	3	2	10	15
4.1 - 5	1	..	10	10
5.1 - 6	..	..	5	10
6.1 - 7	..	..	1	3
Maximum	4.8	4.2	7.2	11.0
Minimum	0.4	0.4	0.8	0.9
Maximum of fibers	44% 0.8-1.2 microns	45% 1-1.6 microns	41% 1.1-1.5 microns	35% 1.3-2 microns

Another way of controlling the exactness of these measurements is a comparison with the size and distribution of the pyramidal tract in the human fetus before myelinization takes place. Such investigations were carried out by Flechsig [10] on human material, and by von Lenhossek,[11] on some mammals. The averages of cases 1 to 10 are compared with their numbers, and, when necessary, are calculated from the second or third cervical as of the value of 100.

It is evident from table 9 that the percentage distribution of the pyramidal tract in the spinal cord is, within narrow limits, the same in the nonmyelinated area of the fetus of 48 cm. as it is in the demyelinated area in lesions of the pyramidal tract in adult man. Furthermore, the

9. Ziehen, P.: Handbuch der Anatomie des Menschen. Anatomie des Zentralnervensystems, Jena, Gustav Fischer, 1899, vol. 4.

10. Flechsig, quoted by Ziehen (footnote 9).

11. Von Lenhossek, M.: Ueber die Pyramidenbahnen im Rueckenmark einiger Säugtiere, Anat. Anz. **4**:208, 1889.

plan of distribution in the new-born cat and mouse very much resembles that in the human fetus and the new-born child as well as in the adult. In the guinea-pig there is a striking deviation within the lower cervical cord. A repeated investigation of a larger amount of material would be of interest.

A comparison of the absolute size and percentage distribution of the pyramidal tract in different cases will naturally be valueless unless one takes cognizance of the degree of condensation of the pyramidal tract fibers within the various segments of the cord. Thus, it becomes necessary to know the number of fibers per square millimeter in order to draw any definite conclusions. This problem of counting the pyramidal tract fibers was difficult. Case 10 and a normal case were selected for this purpose, and Bielschowsky-stained blocks and paraffin-formaldehyde material stained with the rapid modification of the Loyez method and

TABLE 9.—*Segmental Distribution of Pyramidal Tract: Comparative Values*

Segment	Averages of Cases 1 to 10	Flechsig Average Distribution	Averages of Cases 1 to 10	Flechsig New-born Child	Flechsig Fetus 48 Cm.	Averages of Cases 1 to 10	Von Lenhossek		
							Cat	Guinea-Pig	Mouse
C I	...	...	...	...	...	100	100	100	100
C II	...	...	100	100	100	...	...	...	...
C III	100	100	...	...	...	...	...	...	...
C IV – VII	81	87	...	...	...	...	...	...	...
C V	...	...	79	84	87	...	...	...	...
C VII	...	...	...	...	...	63	60	36	54
C VIII	...	...	64	72	66	...	...	...	...
D III	55	62	51	52	52	...	...	...	...
D VI	46	53	41	45	42	39	42	30	34
D X	...	...	34	40	40	...	...	...	...
D XII	35	39	...	...	...	...	...	...	...
L II	...	...	...	...	...	27	33	29	32
L IV	20	24	20	...	23	...	...	...	...

the Weigert-Pal method were used. The technic has already been described. If one always takes care to photograph the same region of the pyramidal tract, i.e., the border zone of inner and middle third, the results will be uniform, as shown in table 7. It seems that there is a slightly larger number of fibers in the upper cervical segments than in the dorsal segments. It is important for the conclusions to be drawn later to note that in the eighth cervical, twelfth dorsal and first lumbar segment the condensation of pyramidal tract fibers is practically the same. In no event are they higher in the dorsal region than in the cervical segments.

A difference of from 20 to 34 per cent between myelinated fibers and axons counted in Bielschowsky preparations has been noted in other material. Ranson [12] described the phenomenon in the fasciculus cerebrospinalis of the albino rat and Linowiecki [13] saw the same difference in

12. Ranson, S. W.: The Fasciculus Cerebro-Spinalis in the Albino Rat, Am. J. Anat. **14**:411, 1913.

13. Linowiecki, A. J.: The Comparative Anatomy of the Pyramidal Tract, J. Comp. Neurol. **25**:509, 1914.

the pyramidal tracts of *Macacus rhesus* and other mammals. Table 8 shows that, under an oil immersion lens, fibers as small as 0.9 micron are stained with the Loyez method, and that most of the myelinated fibers are from 1 to 2 microns thick (from 43 to 54 per cent). The difference between myelinated fibers and Bielschowsky-stained axons, as shown in table 7, is due to the smaller number of myelinated fibers in the range of from 0.3 to 1 micron, as shown in table 8 (45 and 33 per

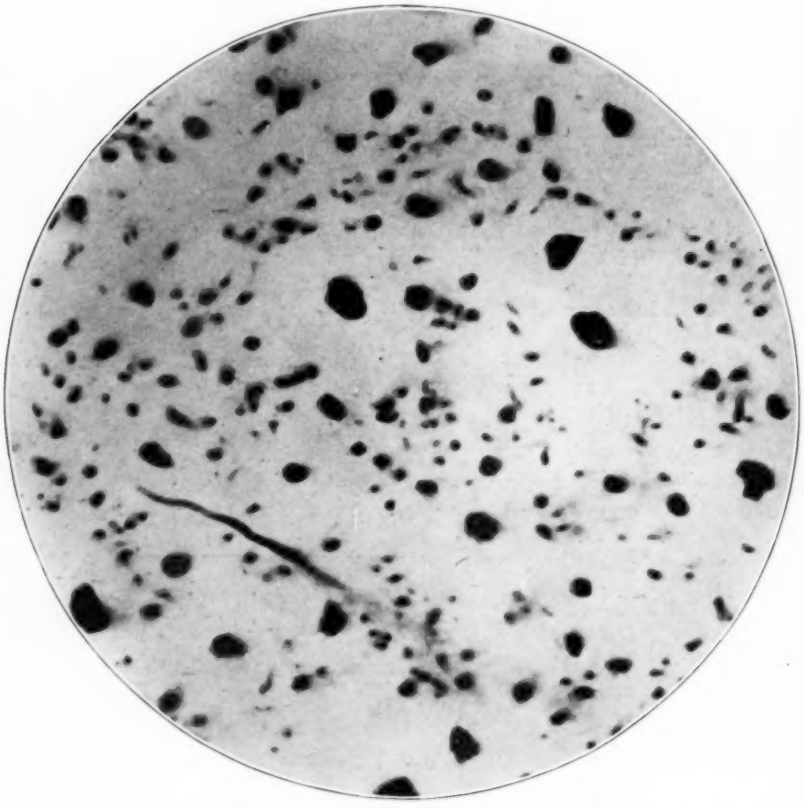

Fig. 1 (case 10).—Transverse section through normal lateral pyramidal tract at the eighth cervical. Bielschowsky stain. Leitz 2 mm. objective, 8 × ocular; linear enlargement, 1,400 ×. Compare with figure 2.

cent difference). This difference between numbers of fibers as estimated from myelin sheath preparations and those by the Bielschowsky method may be seen by comparing figures 1 and 2. In the lumbar region, the relative number of larger fibers is higher than in the cervical segments. This is contradictory to the present assumption that the size of an axon decreases proportionately with the distance from its ganglion cell. This slight discrepancy may be explained by the mixture with other tract

fibers, which is more pronounced in the lumbar than in the cervical region. There is no evidence of a change in the condensation of the axons of the pyramidal tract in their course through the spinal cord. Flechsig [10] gave the size of myelinated fibers in a new-born child as from 2 to 4.5 microns. In comparison with the figures of table 8 this would mean that in the new-born child only larger fibers may be recognized as myelinated. The pale appearance of the pyramidal tracts in Weigert

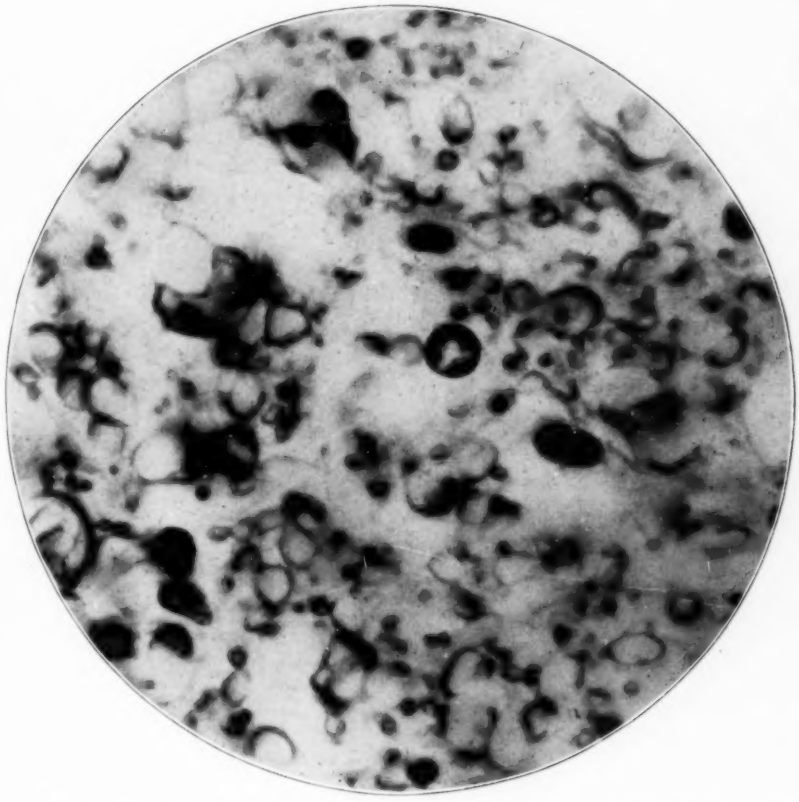

Fig. 2 (case 10).—Section through lateral pyramidal tract at the eighth cervical. Stained with rapid modification of Loyez' method. Optic system same as in figure 1. Compare with figure 1.

preparation of new-born children makes this explanation extremely probable. Flechsig gave the total number of pyramidal tract fibers in the upper cervical region as "considerably more than 100,000." Calculated from measurements in case 10, and by comparison with the normal case, this number should be approximately 500,000 for both right and left lateral tracts, or about 250,000 for the tract of one side. The number may be found to be larger when technical methods have been improved.

Of interest in connection with the question of hemiplegia, which will be discussed later, is the relation between crossed and uncrossed pyramidal tract fibers. Flechsig stated that in the human fetus the crossing is complete in 15 per cent of the cases. In the material here presented, there was evidence of an anterior tract in eight of ten cases. In four of these cases no definite, well formed tract could be made out, but disseminated degenerated fibers were present. In the remaining four cases, the degenerated area of the anterior tract was from 8 to 16 per cent of the size of the lateral tract. This corresponds with Flechsig's figures, which give from 8 to 20 per cent as the relative size of the anterior pyramidal tract in a fetus of about 49 cm. length. Figures from textbooks, which show cases of diplegia with demyelinization of both anterior columns as representative of the position of the anterior tract, are misleading. It may occupy the zone next to the anterior fissure or may be placed more laterally, as shown schematically in Bailey's [14] textbook. In the upper dorsal segments, the trace of anterior pyramidal tract fibers in sections stained for myelin sheaths is lost. These observations as well as the numbers of the relative distribution of the pyramidal tract controvert Bok's [15] opinion that a large part of the pyramidal tract fibers of the lateral funiculus is destined for the supply of the lowest part of the spinal cord; or that the lateral tract always receives a new supply of fibers from the anterior tract.

Figure 3 illustrates graphically the proportions of pyramidal tract fibers which are destined for the supply of the different segments. At the same time it demonstrates the amount of musculature innervated by each segment. The upper broken line represents the average values of the percentage distribution of musculature for six females, and the lower broken line for nine males. The solid line represents the percentage distribution of the pyramidal tract, calculated from the area of degeneration in ten cases of hemiplegia. The rectangles at the base indicate the amount of pyramidal tract fibers which supply 1 per cent of those muscles which are innervated by different segments of the spinal cord. The segments are arranged in five groups according to the different plexuses originating from them: from the first to the fourth cervical segments, cervical plexus; from the fifth cervical to the second dorsal, brachial plexus; from the third to the eleventh dorsal, thoracic nerves, and from the twelfth dorsal to the fourth lumbar, lumbar plexus; from the fifth lumbar to the fifth sacral, sacral plexus. It is evident that the muscles innervated by the first four cervicals are best supplied with

14. Bailey: Textbook of Histology, edited by O. S. Strong and A. Elwyn, ed. 7, New York, William Wood & Company, 1927.

15. Bok, S. T., in von Möllendorff, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1928, vol. Nervensystem, part 1, p. 562.

pyramidal tract fibers; the segments from the third to the eleventh dorsal range next; from the fifth cervical to the second dorsal are third, and from the twelfth dorsal to the fourth sacral range fourth, with a slight difference in favor of the fifth lumbar to the fourth sacral.

This difference in the distribution of pyramidal tract fibers becomes highly interesting if one compares it with the function of the corresponding muscle groups, as present in lesions of the pyramidal tract. Thomas [16] gave an illustrative description of their function in hemiplegia, at the same time summarizing older reports. He quoted the classic observations of Broadbent in 1866. Broadbent pointed out that the muscles which escaped paralysis in hemiplegia are those which act more

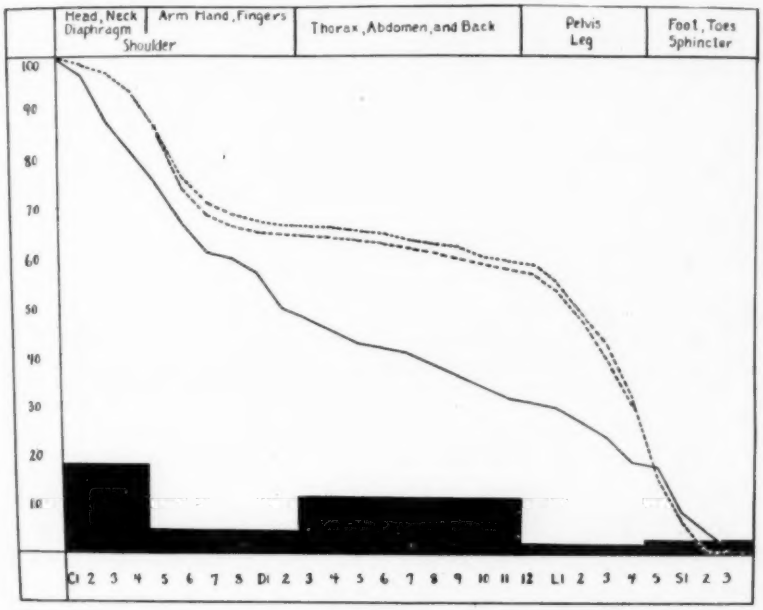

Fig. 3.—The upper broken line represents the averages of six females; the lower broken line, the averages of nine males. Both indicate the percentage distribution of musculature in relation to segmental innervation. The solid line represents the percentage distribution of the pyramidal tract at the different segments of the spinal cord. The area at first cervical segment equals 100. The black rectangles show the percentage of pyramidal tract fibers which innervate 1 per cent of the musculature. The numbers at the ordinate indicate percentages. They should be divided by five in order to obtain the exact numbers for the black rectangles.

or less constantly in association with corresponding muscles of the opposite side of the body. He considered as entirely bilaterally acting muscles those of the eyes, the back, the neck and the chest. Therefore,

16. Thomas, K. M., in Osler: Modern Medicine, Philadelphia, Lea & Febiger, 1928, vol 6, p. 377.

movements of respiration and forward and backward movements of the trunk are not affected. To explain these facts Broadbent advanced the theory that the spinal nuclei corresponding to these muscles are closely associated. Thomas, however, pointed out that this simple theory could not explain all the clinical facts. He assumed an association between the two cerebral centers themselves without going into detail concerning this association. Foerster,[17] who studied the difference in the paralysis of the various muscle groups in the paralyzed extremity, came to the conclusion that the Wernicke-Mann type of predilection really existed in hemiplegia. In order to explain these differences in voluntary innervation of different functions in the paralyzed extremity, he advanced the theory that homolateral uncrossed fibers of the pyramidal tract (Hilfsfasern), originating in cortical centers of the homolateral hemisphere (Hilfsursprungsfeld), innervated the nuclei of the muscles in question. In the case of destruction of the crossed pyramidal tract they assume the function of the main tract. Foerster did not deal, however, with the nonparalyzed muscles of the trunk, thorax and abdomen.

In studying figure 3 it becomes obvious that the two regions which are best supplied by pyramidal tract fibers correspond to muscle groups which are not paralyzed in apoplexy: from the first to the fourth cervical segments, mainly for the muscles of the neck, and partly for the diaphragm; from the second to the twelfth dorsal, for the muscles of the thorax, the abdomen and the back, or, speaking in physiologic functions, for respiration and movements of the trunk. The corresponding numbers for the supply of the upper extremity are only one-fourth, those for the lower extremity only one-sixth, of the figures for the first four cervical segments and one-third and one-fourth respectively of the figures for the thoracic segments.

Foerster's assumption that pyramidal tract fibers originating in the homolateral hemisphere run in the anterior pyramidal tract, or as uncrossed fibers of the homolateral lateral tract, is not supported by the anatomic observations. In Marchi preparations of early cases of hemiplegia one finds only scattered degenerated fibers, or none, in that lateral pyramidal tract of the spinal cord which arises from the normal hemisphere. In other words, nearly all of the fibers of the lateral tract have crossed in the decussation. Furthermore, in only 50 per cent of all cases is there found a well developed anterior pyramidal tract while in 20 per cent this tract is absent entirely. The size of this tract is from 8 to 16 per cent of the size of the lateral tract. It ends in the upper thoracic segments and its fibers cross to the opposite side in their course through the spinal cord. To correlate the great number of pyramidal

17. Foerster, O.: Ueber den Laehmungstypus bei corticalen Hirnherden, Deutsche Ztschr. f. Nervenh. **37**:349, 1909.

tract fibers supplying nonparalyzed muscles in hemiplegia with the theory of bilateral innervation, it would be necessary to assume that the homolateral fibers take their course together with the crossed pyramidal tract; further, that they cross in the decussation and that they have a second crossing at the approximate level of the segment of the spinal cord which they innervate. Such a crossing within the spinal cord is assumed for the anterior pyramidal tract fibers, and a crossing and recrossing is well known in other fiber systems, e.g., in the anterior spinocerebellar tract. Only such an accessory hypothesis would explain all the facts which were found in the preceding investigations, namely: 1. There is no difference in the percentage distribution of the degenerated area throughout the spinal cord, whether one is dealing with a cortical or a capsular lesion. 2. The degenerated area of the lateral tract is confined to the side of the spinal cord opposite to that of the lesion. 3. The number of fibers supplying the nonparalyzed muscle groups is from three to six times as large as those supplying the extremities. All such deductions naturally are based on the main hypothesis that voluntary innervation of striated muscles takes its way solely from the precentral gyrus by the pyramidal tract.

The question which arose at the beginning of this investigation was as follows: Are the muscles of the hand in man better supplied with pyramidal tract fibers than are other muscles? Foerster's direct electric stimulation experiments indicate the seventh cervical to the first dorsal as the main area of innervation of the muscles of the hand. The figures for the weight of muscles innervated from these segments and for the relative part of pyramidal tract fibers are the following:

Weight of Muscles		Percentage of Total Muscles		Percentage of Pyramidal Tract	Percentage Pyramidal Tract Innervating 1 per Cent Muscle	
Male	Female	Male	Female		Male	Female
1,011	752	8.3	9.0	11.0	1.3	1.2

The figures of the last two columns are nearly the same as calculated for the total musculature supplied by the fifth cervical to the second dorsal; or, in other words, there is no evidence of a better supply for the muscles of the hand than for other muscles of the body. Further investigations will show whether various animals have a different scheme of pyramidal tract innervation of the extremities.

CONCLUSIONS

1. In ten cases of hemiplegia with various anatomic lesions the area of the degenerated pyramidal tract in the spinal cord was measured, and the segmental distribution of fibers was calculated.

2. The results show that approximately 50 per cent of all pyramidal tract fibers supply the cervical, 20 per cent the dorsal and 30 per cent the lumbar segments.

3. This scheme of distribution is the same in human adults as in new-born children and in new-born animals in which the area of the unmyelinated pyramidal tract has been measured.

4. The weight of male and female muscles and the absolute and relative part of the musculature innervated by different segments of the spinal cord were calculated from the combined tables of Theile's investigations on human muscle and Foerster's tables of segmental innervation, as deduced from electrical stimulation, Bing's figures being used as second choice.

5. The cervical segments innervate approximately 33 per cent of the total musculature, 10 per cent is innervated by the thoracic segments and 57 per cent by the lumbar segments.

6. The percentage of the total pyramidal tract fibers innervating a unit of muscle (1 per cent) within a certain group of segments was calculated. The results are as follows: first four cervical, 3.7; fifth cervical to second dorsal, 1; third to eleventh dorsal, 2.4; twelfth dorsal to fourth lumbar, 0.5; fifth lumbar to fourth sacral, 0.6.

7. This relative distribution demonstrates that the segments which innervate the musculature of the neck and trunk receive from three to six times as many fibers as those which innervate the extremities. It should be noted in this connection that the muscles of the neck and trunk are not paralyzed in hemiplegia.

8. The muscles of the hand are not better supplied by pyramidal tract fibers than are the rest of the muscles of the arm or of the shoulder. The upper extremity as a whole receives approximately double as many fibers per unit of muscle weight as the lower extremity.

9. In two of the ten cases investigated no evidence of an anterior pyramidal tract could be found; in four cases only scattered degenerated fibers in the corresponding anterior column were found, while in four cases there was a well defined area of degeneration measuring from 8 to 16 per cent of the area of the degenerated lateral tract.

THE ORIGIN OF OLIGODENDROGLIA

DATA DETECTED IN TUMORS OF THE BRAIN *

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The origin of the oligodendroglia is still a debated question. This cell, which Cajal called the "third element" of the brain, apparently has an origin different from that of the microglia. Del Rio Hortega is an advocate of the mesodermic origin of the microglia, although his view is not yet entirely accepted. With the new conception of the ectodermic origin of the meninges, the question of the embryologic derivation of the microglia again comes to the fore and will certainly be the subject of new investigations. I may anticipate my observations by the statement that in my investigations of the meningiomas I have found numerous microglia cells in the tumors themselves. This fact might be considered as in support of the mesodermic origin of the microglia, as well as an argument against it if other elements of ectodermic origin are to be found in the same tissue.

In regard to the oligodendroglia, it has been generally believed that it is derived from the ectoderm, but no proofs either of embryologic or of pathologic nature have been brought forward to uphold this view. The result is that in some of the most recent works on tumors in which this question is discussed, the origin of these cells has been left undetermined (Roussy [1]). Bailey and Cushing [2] show, in their table setting forth the origin of neuroglia, that oligodendroglia is derived directly from the medulloblasts with no indication of intermediary stages.

In an investigation of a tumor of the brain of the glioma group which I have carried out in collaboration with Dr. Davidoff,[3] we have been much impressed by the polymorphism of the tumors belonging to the group of so-called spongioblastoma multiforme. We have emphasized this point and have stressed the fact that in the same tumor numerous areas are found in which the histologic features differ because of differences between the predominating types of cells. Indeed, close to areas

* Submitted for publication, Dec. 21, 1928.

1. Roussy, G., abstract of Bailey: Histological Atlas of Gliomas, Ann. d'anat. path. **5**:801 (June) 1928.

2. Bailey, Percival; and Cushing, Harvey: A Classification of the Tumors of the Glioma Group on a Histological Basis, Philadelphia, J. B. Lippincott Co., 1925, pp. 175.

3. Davidoff, L. M., and Ferraro, A.: Brain Tumors Among State Hospital Patients, Am. J. Psychiat. **8**:599 (Jan.) 1929.

in which the spongioblastoma characters of the tumor are definite areas are seen in which ependymal cells predominate, and others in which astroblasts or astrocytes form the main bulk of the tumor. It follows that practically all stages of development of glia cells can be encountered from the medullary epithelium to the almost adult type of cell. The rarity, however, of areas of so-called oligodendroglioma has rendered the investigation of the origin of this element difficult. It must be remembered here that even Cushing and Bailey, who first spoke of

Fig. 1.—Tumor of the pontocerebellar angle (normal size).

oligodendroglioma, a tumor which corresponds to the "gliome à petites cellules" of Roussy and Lhermitte, had not succeeded, at the time of publication of their book, in impregnating these cells with the specific silver method.

I recently had the opportunity to study an interesting tumor which Dr. A. Saccone, pathologist of the Metropolitan Hospital, was kind enough to let me investigate (fig. 1). The tumor was found occupying the pontocerebellar angle and looked, macroscopically, like the so-called

neurinomas of the acoustic nerve. Dr. Saccone stated that he has been unable to find any connection of the growth with the brain tissue itself, although such a connection may have escaped his attention.

I studied the tumor histologically, and found it to be one of glial nature in which, however, no definite diagnosis in regard to type could be made. It is surprising how different the structure appeared in various areas of the same neoplasm. I found areas of typical spongioblastoma and others in which either ependymal cells or astrocytes predominated. The tumor must be classified, therefore, among the so-called mixed tumors of the glial group.

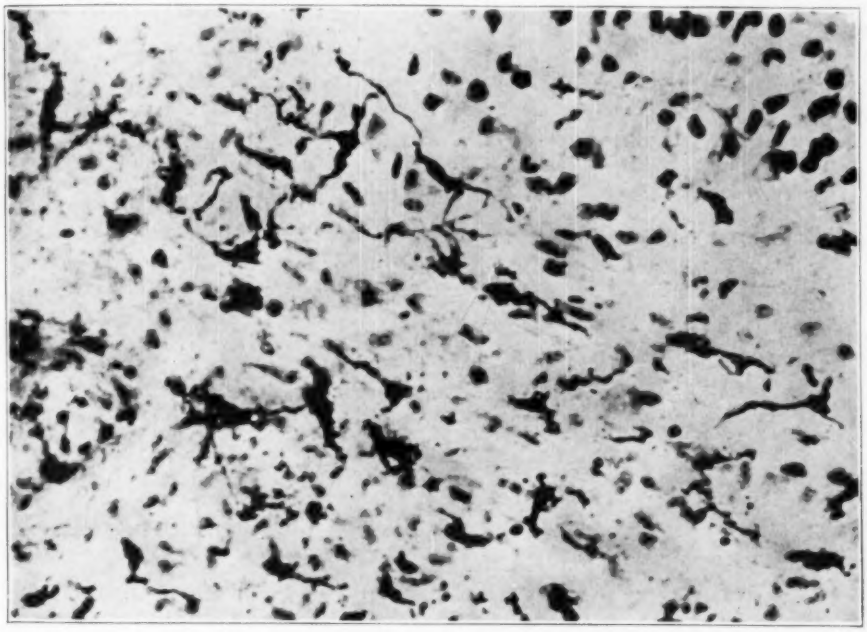

Fig. 2.—Microglia cells in a well preserved area of the tumor. Some of the cells show the pseudopodic appearance described by Del Rio Hortega. Silver carbonate impregnation; × 290.

The presence of a pure glioma in the pontocerebellar angle is a rarity, and its interest lies in the fact that such growths are considered, especially in France, as belonging to the group of glial tumors in the same category with the so-called meningiomas or meningoblastomas. Although in Dr. Saccone's case no gross connection with the remaining central nervous tissue was reported, I feel that it might have existed and have been torn out in the process of removing the brain which unfortunately was destroyed immediately after removal. This impression is based on the fact that one edge of the tumor was covered by a layer

of the lateral recess of the fourth ventricle from which the tumor may have arisen and from which it may have been extroflected into the pontocerebellar angle. Thus, it is possible that some of the tumors of the pontocerebellar angle may have an origin other than that of perineural sheaths.

HISTOLOGY OF THE TUMOR

The study of this tumor has yielded the following data concerning the histology of the gliomas:

1. Concerning the presence of microglia in the tumor itself, Penfield [4] has already drawn attention to the fact that microglia cells are to be

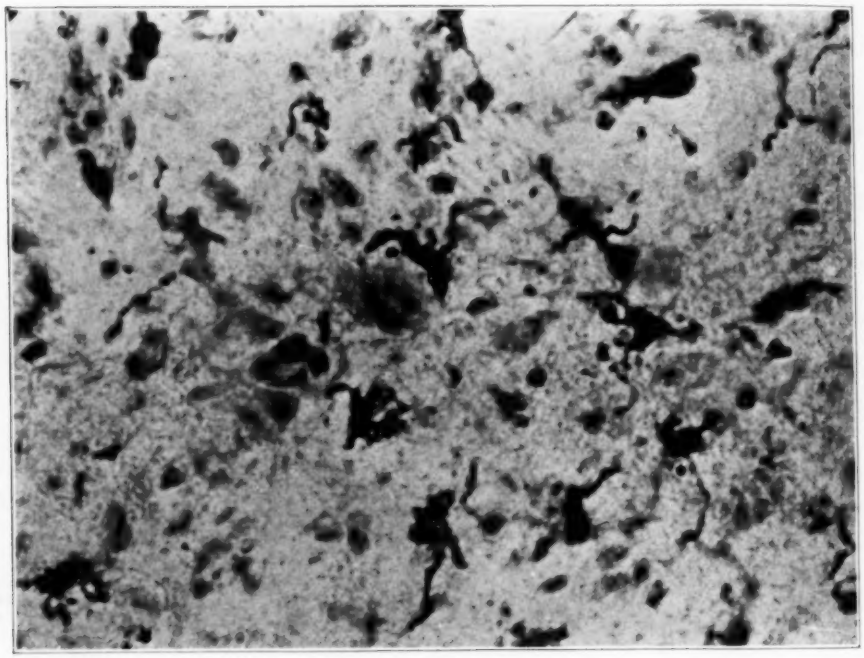

Fig. 3.—Microglia cells. Pseudopodic appearance of the cells in a well preserved area. Del Rio Hortega silver carbonate impregnation; × 290.

found within gliomas as well as at their periphery. "Within the tumor the microglia discharges the function of dendrophagocytosis, while in the nervous tissue surrounding the tumor these cells discharge the function of phagocytosis of the products of cerebral destruction." Penfield never found microglia in well vascularized areas of the tumor.

Cushing and Bailey have seen microglia cells extend only as far as the boundary of the neoplasm but have not identified them definitely

4. Penfield, W.: Microglia and the Process of Phagocytosis in Gliomas, Am. J. Path. **1**:77, 1925.

as neoplastic cells. In one case only did they obtain an impregnation of the tumor cells by Hortega's method for staining the microglia. Against their microglial nature, however, they urge: (*a*) that the processes do not show the lateral excrescences characteristic of microglia cells; (*b*) that the processes of some of the cells are attached to the vessel walls in a manner similar to sucker feet, and (*c*) that the tumor is but an isolated example with no confirmatory evidence.

In my case, the presence of microglia cells in the tumor itself was undoubted. There were even areas in which the predominating cells were microglia. That some of these cells were transforming into com-

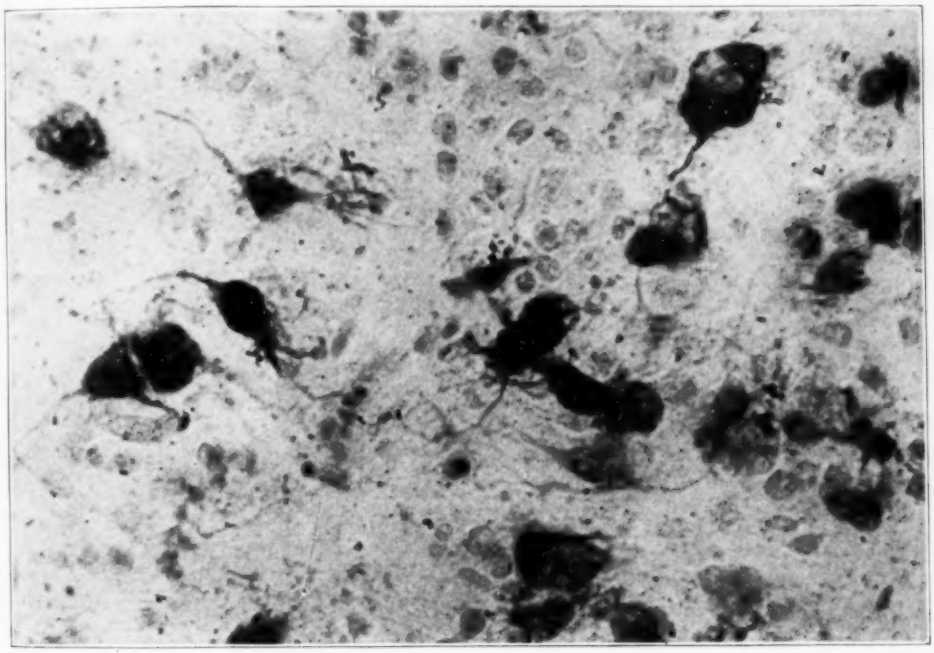

Fig. 4.—Oligodendroglia cells, isolated or collected in clusters. Silver carbonate impregnation; × 580.

pound granular corpuscles is also certain, since all the intermediate stages between normal cells and reticulogranular cells were found. This was seen within the tumor itself in areas undergoing degeneration. In some areas, however, in which no signs of degeneration were seen, normal microglia cells were found. In other well preserved areas the microglia elements disclosed the peculiar "pseudopodic" appearance which Del Rio Hortega described in the immature stages of their development (figs. 2 and 3). This fact seems important to me, as the presence of microglia cells in an embryologic stage points out the neoplastic nature of these

elements and would justify the use of the term microglioma for this particular area.

2. Another interesting observation was the presence in the same tumor of numerous oligodendroglia cells which were at times isolated and at others collected in clusters. The cells were unusually large, and at first glance under low power manipulation they gave the impression of astrocytes. The elements were, however, typical oligodendroglia cells, with a more or less round body, round nucleus and few prolongations. Under a higher magnification the large cells appeared to resolve into

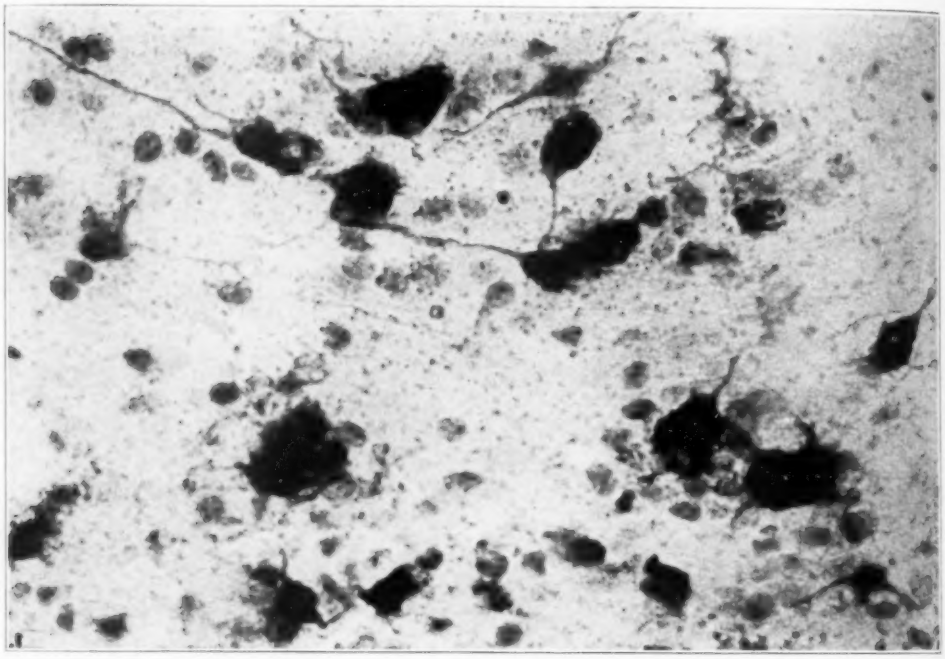

Fig. 5.—Oligodendroglia cells isolated and collected in clusters. Silver carbonate impregnation; × 580.

a collection of smaller elements, some of which were spongioblasts and others oligodendroglia cells which could be impregnated with both silver carbonate (figs. 4 and 5) and gold sublimate.

The fact that these cells were found scattered here and there is also an interesting point, since it shows that oligodendroglia cells are to be found not only in areas in which tumor shows the character of an oligodendroglioma. Indeed, in this particular tumor no such definite areas were found. The number of oligodendroglia cells was, however, so high and the size of some of them was so unusual that it seems safe to admit their neoplastic nature.

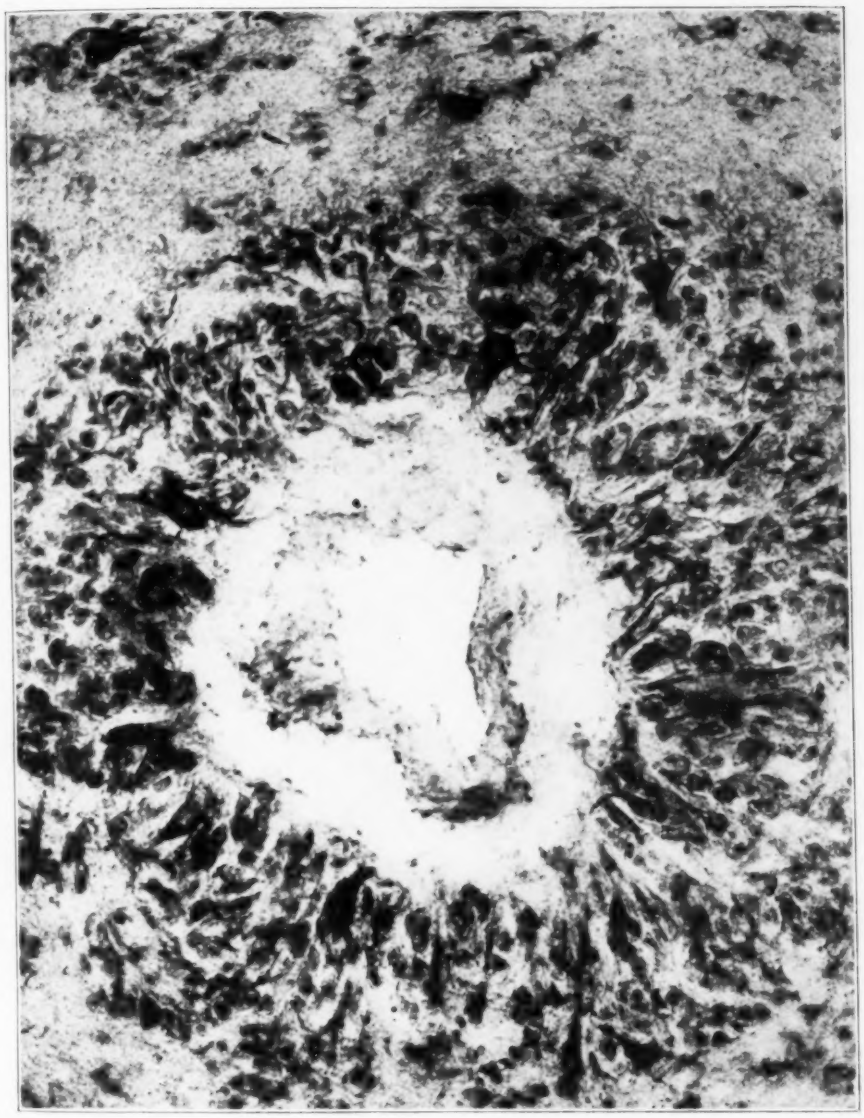

Fig. 6.—Spongioblasts among which transitional stages in the formation of oligodendroglia cells are to be seen. Gold sublimate method; × 300.

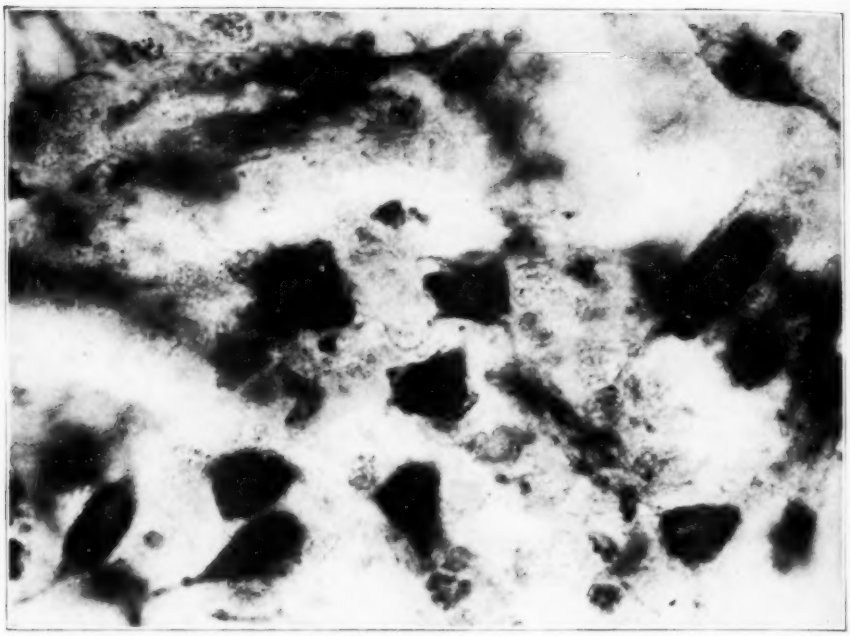

Fig. 7.—Oligodendroglia cells among spongioblasts in a spongioblastoma multiforme. Gold sublimate impregnation; × 665.

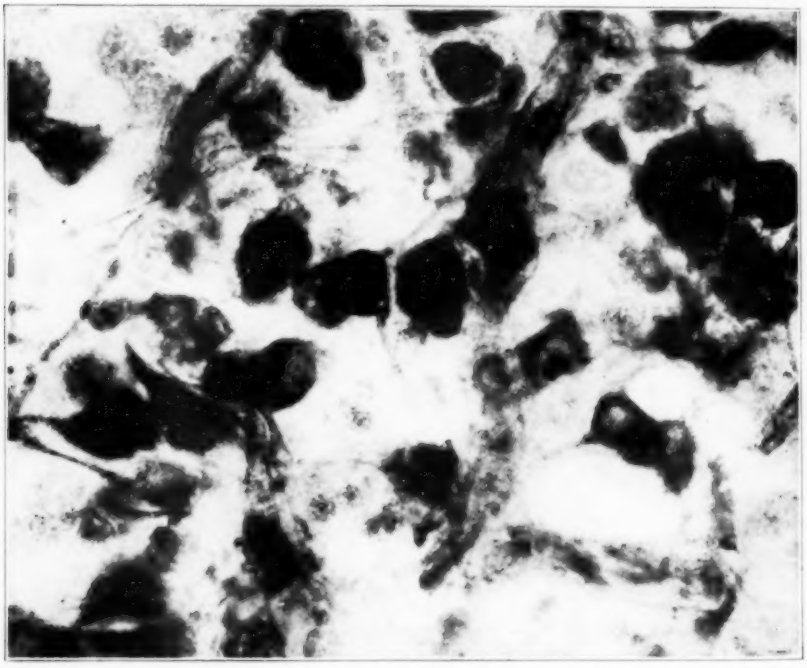

Fig. 8.—Transitional stages between spongioblasts and oligodendroglia cells. Gold sublimate method; × 665.

I must here emphasize the fact that the oligodendroglia cells which I have found scattered through the sections showed, at times, transitional stages in the form of the astrocyte. This fact, which is also mentioned by Penfield, has always attracted my attention and seems to me important, since it points to a common origin of both, in connection with the fact that oligodendroglia cells transform into compound granular corpuscles. The study of this particular tumor confirms the conclusions of my previous investigations in collaboration with Dr. Davidoff [5] as to the reaction of oligodendroglia cells to injuries of the brain. This transformation takes place particularly in the areas undergoing degenera-

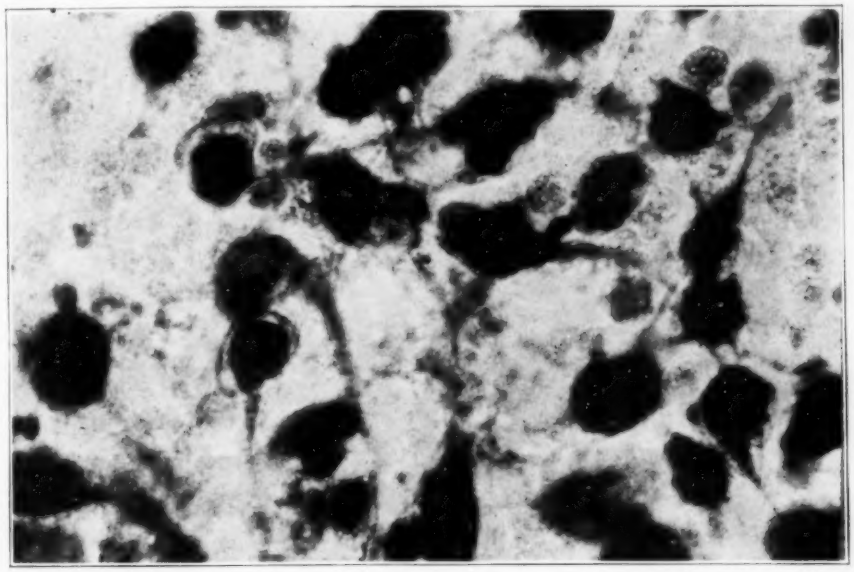

Fig. 9.—Acute swelling of cells (spongioblasts and oligodendroglia) in a spongioblastoma multiforme. Gold sublimate impregnation; × 665.

tion, and all the transitory stages can be followed, from oligodendroglia to compound granular corpuscles.

3. The last point which I shall discuss in connection with this tumor is the origin of the oligodendroglia cells. In various parts which I have considered as areas of spongioblastoma, among the cells of the spongioblastic type were to be seen a certain number of other elements which, on careful study, proved to be oligodendroglia cells. Their presence among the spongioblasts brings up the question of their possible derivation from these elements, and I have searched for transitional stages between these two elements. The result of this investigation justifies

5. Ferraro, A., and Davidoff, L. M.: The Reaction of Oligodendroglia to Brain Injuries, Arch. Path. **6**:1030 (Dec.) 1928.

my belief that oligodendroglia cells are derived from a transformation of the secondary spongioblasts.

Figure 6 shows one of the areas in which, among numerous spongioblasts, elements resembling oligodendroglia cells can be seen. The same elements, always showing the characteristic form and structure of

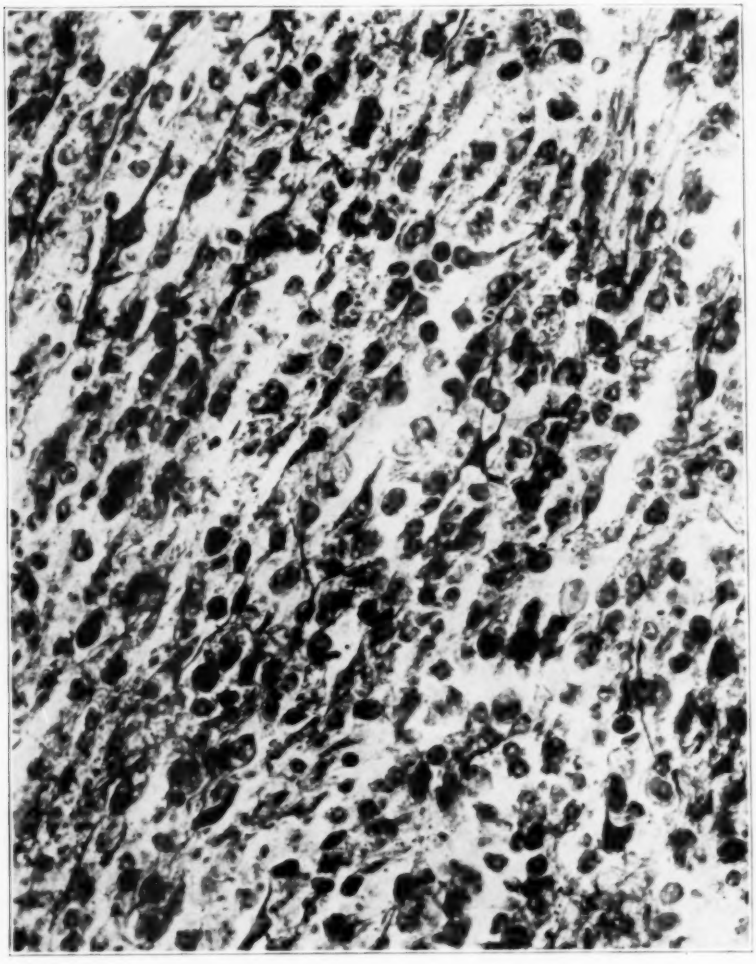

Fig. 10.—Area of oligodendroglioma in a spongioblastoma multiforme. Gold sublimate impregnation; × 215.

oligodendroglia cells, are also to be found scattered through the section in the vicinity of these areas. These investigations have been carried out not only in the case here reported, in which I have been successful with the specific stain for oligodendroglia, but also on a typical spongioblastoma of Dr. Davidoff's collection which he kindly let me study. In this

case the gold impregnation, which was made on material fixed in solution of formaldehyde, was successful and extended to all the spongioblasts. Among all these cells transitional stages could be followed. Some of these are shown in figures 7 and 8. I have no doubt as to the oligodendroglial nature of some of the cells which are reproduced in the figures. Some of them even show the peculiar type of swelling which was first described by Penfield and Cone (fig. 9).

I have been fortunate in finding in the tumor from Dr. Davidoff's collection an area in which the majority of cells had the typical columnar arrangement of the cells seen in the oligodendroglioma (fig. 10). Spongioblasts were found among these cells, and this fact in connection with the close coexistence of areas of oligodendroglioma and areas of spongioblastoma is a still further argument that a close relationship exists between the spongioblasts and the oligodendroglia cells.

The histologic details of these two cases have led me to the conclusion that oligodendroglia cells arise from the secondary spongioblasts, that is, from the unipolar cell through transitional stages during which prolongations are developed while the cell body acquires a more rounded aspect and the nucleus a better outline, often with a more deeply stained periphery.

CONCLUSIONS

A pontocerebellar tumor is described, the microscopic structure of which was that of a mixed tumor of the glioma group. Areas were found in which spongioblasts predominated and others in which the astrocytes, the ependymal cells, the oligodendroglia and the microglia respectively, formed the chief elements.

The presence of a glioma in the pontocerebellar angle is not common, and its interest lies in the fact that some recent investigators have asserted that all the tumors of the meninges, as well as those of the pontocerebellar angle (usually acoustic neurinomas), are of ectodermic rather than of mesodermic origin.

The presence of microglia cells in the tumor itself in areas free from disintegration supports the view that these elements may form a tumor mass.

The presence of numerous oligodendroglia cells in clusters of various sizes is interesting since the case is one of the few in which specific impregnation of oligodendroglia cells has been successfully reported. The importance is increased by the fact that oligodendroglia cells not only are found in areas of definite oligodendroglioma but are scattered in the glioma itself. This upholds the point that Davidoff and I raised in our previous paper as to the presence of oligodendroglia cells in other types of gliomas. Oligodendroglia cells are seen transforming into compound granular corpuscles.

The origin of oligodendroglia cells is discussed, and facts are brought forward in favor of the derivation of the cells from the spongioblasts.

THE EARLY DIAGNOSIS OF MENTAL DEFECT*

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The process of growth itself, whether normal or defective, is beyond the eye of immediate perception. The symptoms of growth, however, can be studied with direct objectivity. Psychologically, growth expresses itself in ascending orders of behavior. The trend and the tempo of development in a human being can be deduced from the manner in which capacities for behavior mature. The possibility of diagnosing developmental types of mental defect in early infancy rests on certain lawful relations between age and growth.

It takes time to grow. Development is profoundly conditioned by duration. Although one cannot say that development is caused by the clock or the almanac, one may insist that development is a function of age, and that a recognition of the age values of behavior and of the behavior values of age constitutes the first essential for developmental diagnosis. The frequent failure to discern certain kinds and grades of mental defect in early infancy may well arise out of the fact that precise heed has not been paid to the factor of agedness in appraising the young infant.

In early infancy it is necessary to give much respect to small units of duration. The younger the organism the more potent is any given unit of time in the economy of its development. The younger the organism the more swift and intense its growth tends to be. In the period of the germ and embryo, hours and days count; in the period of the fetus, weeks; in infancy, months, and in childhood, years. The intense compression of development in the early sector of the life cycle is so great that age, by virtue of some kind of relativity, assumes an augmented importance which must be reckoned with in diagnostic procedure. Indeed, it may almost be said that in infancy the months are as vast as later years.

The older the child the longer it takes for him to grow a definite proportional amount. This fact may be represented in a diagram (chart 1). By expressing developmental maturity in terms of developmental age, it is possible to plot the diminishing increments of development which accompany aging on a basis of relative percentage. Chart 1 shows the equivalence of eleven monthly increments of developmental age prior to the first birthday, and eleven corresponding yearly incre-

* Submitted for publication, Jan. 31, 1929.

ments thereafter. By using the convenient concept of the developmental quotient, this chart brings out the genetic relativity of the factor of age.

It is this relativity which has some significance for the diagnosis and the portrayal of mental growth in early infancy. The chart simply brings each interval of agedness into comparison with the total preceding interval. There is a rapid stepping down of increments. In the second month the infant doubles his age; there is a gain or "increment" of 100 per cent. In the third month the increment is 50 per cent (3 months: 2 months). In the fourth month it is 25 per cent (5 months: 4 months). And then in order it is 20, 16.66, 14.28, 12.5, 11.11, 11 and 9.09 per cent, when the first year of the development cycle is complete.

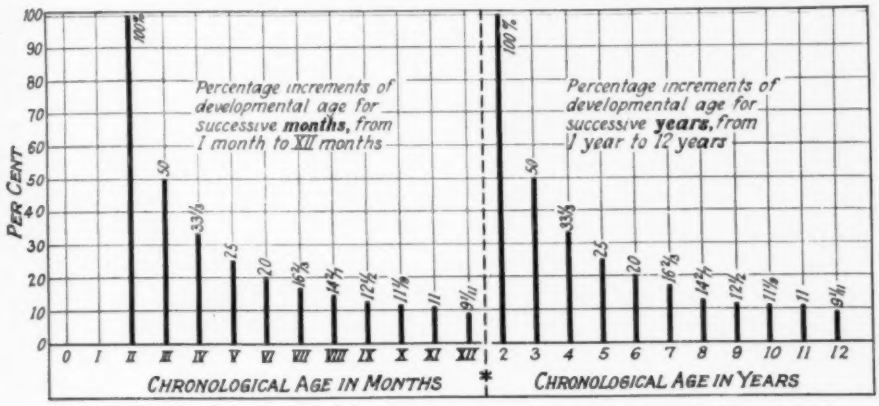
PER CENT
Percentage increments of developmental age for successive months, from I month to XII months
Percentage increments of developmental age for successive years, from 1 year to 12 years
100%
50
33⅓
25
20
16⅔
14⅔
12½
11⅑
11
9⅑
0 I II III IV V VI VII VIII IX X XI XII
2 3 4 5 6 7 8 9 10 11 12
CHRONOLOGICAL AGE IN MONTHS
CHRONOLOGICAL AGE IN YEARS

Chart 1.—Showing the equivalence of eleven monthly increments of developmental age, prior to the first birthday, and, for comparison, eleven corresponding yearly increments thereafter.

It is necessary now to shift to larger units of agedness, or linger on a tedious plateau. A shift to year units, and one has a series of annual increments which arithmetically correspond to the monthly increments. In the second year, the child doubles his age. The series of percentage increments to the thirteenth year is 100, 50, 33.33, 25, 20, 16.66, 14.28, 12.5, 11.11, 11 and 9.09.

The beginnings and the end of the developmental span are extremely inaccessible, so I have simply brought into diagrammatic comparison or analogy the monthly increments of the first year of postnatal life with the annual increments thereafter to the teens. One finds that they are alike, and is left with the broad suggestion that any year of development after the first birthday to the teens is comparable in its relative maturational value to a corresponding month in the first year of life.

From a clinical point of view, such a comparative formulation may well be defensible. Clinical experience surely justifies the general statement that the younger the child the more serious is the prognostic significance of every degree of true retardation. Retardation, like a shadow, lengthens with the lapse of time.[1]

In the early phases of the mental growth cycle, the dynamic importance of small units of time is so great that every month witnesses significant increments of behavior. This process of progressive incrementation begins amazingly early, the nervous system being conspicuously in the van during intra-uterine growth. As early as the second fetal month, rudimentary reflexes appear in the embryo; in the third month, mouth movements; in the fourth month, deep cervical reflexes involving arm, leg and head; in the fifth month, labyrinthine reflexes of equilibrium; in the sixth month, prerespiratory reflexes; and in the seventh, eighth and ninth months, the circumnatal reflexes necessary for extra-uterine existence—a prudential provision against the contingency of premature birth.

By the close of the first postnatal month the normal infant is already showing selective regard for his mother's face. In another month he follows her moving figure with his eyes. At the third month he can crudely manipulate a rattle. At the fourth month he closes in on a toy dangled above his crib. At 5 months of age he can pick up the toy on manual contact. At 6 months he reaches for it on sight. At 7 months he can scoop up a tiny pellet from the table. At 8 months he looks for a spoon fallen out of sight. At 9 months he combines a cup and spoon in play. At 10 months he plucks a pellet with precise pincer prehension. At 11 months he pokes a rod in a hole. At 12 months he articulates one or two words.

Such behavior events are the symptoms of mental growth. They occur, not hit or miss, but in lawful accordance with the time-conditioned mechanics of development. By taking critical account of this biologic relationship between these small units of age and ordered units of behavior, one finds it possible to appraise the normality of tempo and trend in the infant's early development.

Toward this end, I have drawn up a graded syllabus of 135 items of behavior covering the first year of mental growth—approximately a dozen items for each of the twelve successive months. This syllabus has been codified into a normative schedule for the diagnosis and interpretation of developmental status in infancy. By means of the schedule, it is possible to make objective clinical estimates of mental retardation and deficiency in early life.

1. Adapted from Gesell, Arnold: Infancy and Human Growth, New York, The Macmillan Company, 1928, chapter 7.

A large number of children, both normal and subnormal, have now been clinically examined with the aid of this monthly increment schedule and the preschool developmental norms. Some of the cases have been followed for a period of years, and it appears that retardation in the first year is not readily outgrown, but tends to project itself proportionately into the subsequent periods of life. This can be demonstrated by a brief review of the histories of mental growth of a few illustrative studies of cases.

The cases are selected because they are typical of simple, primary forms of developmental retardation. It is clear that sweeping generalizations concerning mental deficiency must be avoided. There are more or less atypical growth complexes which show deceleration, irregularity, deterioration and compensatory acceleration in the course of mental growth. In doubtful and complicated cases, reliance cannot be placed on a single determination of developmental status. The diagnosis must be built up through repeated periodic examinations. The consistency of the series of observations then shapes the prognosis.

TABLE 1.—*Relative Measurements of Developmental and Chronologic Age in Case 1*

Examination	1	2	3	4	5	6	7	8	9
Developmental age level (in months).....	3	4	4.5	7	8	10	13	15	24
Chronologic age (in months).....	3.5	5	6	9	10	12	15	18	30

REPORT OF CASES

CASE 1 (chart 2).—B. C., a boy, was examined nine times between the ages of 3 months and 30 months. The observations in these examinations are summarized in table 1. It will be noted that there was a slight retardation on each of the examinations. The ratings are clinical appraisals expressed as developmental age levels in solar months. (Sometimes fractional and interpolated values are given to express the shadings of the clinical estimate.[2])

The chief question in this and similar histories of growth concerns not the precise accuracy of the individual ratings, but the general consistency of a series of clinical estimates. In this instance there can be little doubt about the general constancy of the observations. Nine is a fairly large number of examinations, and no less significant than an equal number of examinations spread over a period of several years in later childhood. This boy has maintained a steady pace of development consistently pitched on a low average level. He is not mentally deficient. Although the outlook is normal, it is improbable that he will rise to a full average (chart 2).

2. Further details concerning these cases and a discussion of the measurement and prediction of mental growth may be found in Gesell, Arnold: Infancy and Human Growth, New York, The Macmillan Company, 1928. Norms and methods of developmental diagnosis are outlined in Gesell, Arnold: The Mental Growth of the Pre-School Child, New York, The Macmillan Company, 1925.

Case 2 (chart 3).—C. D., a girl, was examined six times between the ages of 9 months and 4 years. Her chart presents a consistently subnormal tempo and trend of development throughout this whole period. Clinically she must be classified in the borderline group. On the first examination, at the age of 9 months, her personality made an indelible impression of amenability, good nature and sociability. Her temperamental make-up from the beginning has been as characteristic of the behavior picture as has her modest mentality. It is possible, but not probable, that this behavior picture will materially change with the advancing years.

Case 3 (chart 4).—V. W., a boy, was examined nine times between the ages of 6 months and 7 years. His grade of subnormality dropped definitely to the clinical level of mental deficiency. The retardation remained consistently low,

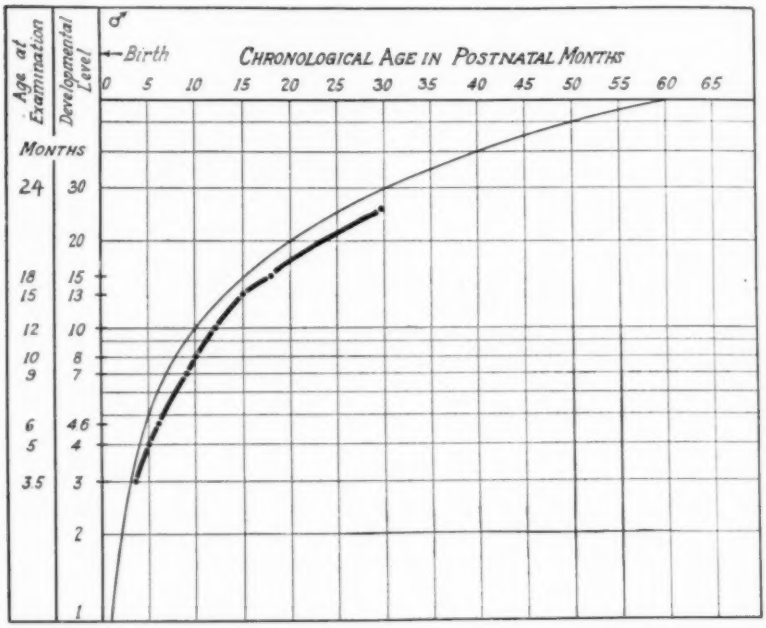

Chart 2 (case 1).—Showing a consistently low average rate in the mental growth of B. C.

as shown by chart 4, with the exception of one clinical estimate made at the age of 12 months. He was then in wretched physical condition, and an unduly liberal allowance was made for this physical handicap. Since that time he had acquired a sturdy physique; but there was no compensating improvement of mental status. That status was one of well defined defect combined with favorable personality and relatively good practical judgment. The mental defect was both inherent and manifest in the behavior picture when he was only ½ year old.

From table 3 it is evident enough that the absolutely small degrees of constitutional retardation in early infancy have considerable relative weight. The inner dynamics of the growth complex are such that, as this typical instance reveals, a retardation of 2 months at the age of ½ year may lengthen into a retardation of approximately 2 years at the age of 6.

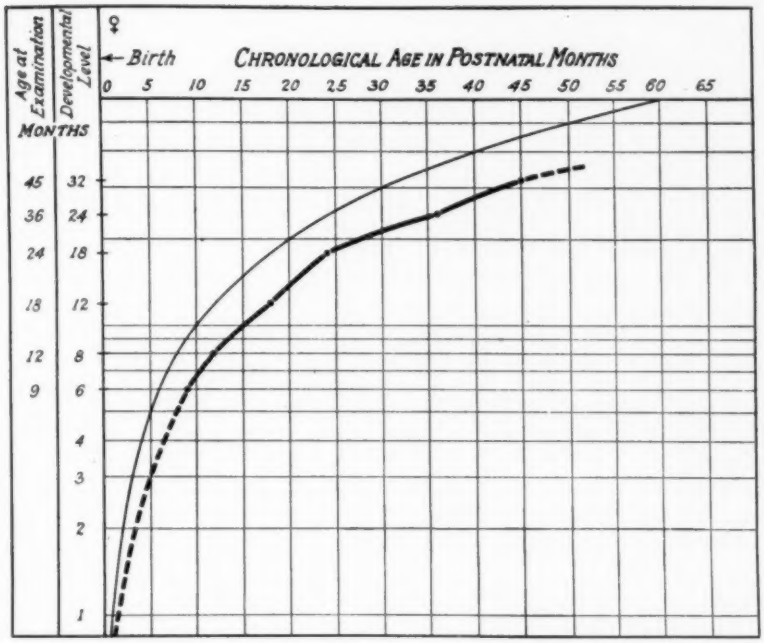

Chart 3 (case 2).—Showing a consistently subnormal development in the mental growth of C. D. from early infancy to the age of 4 years.

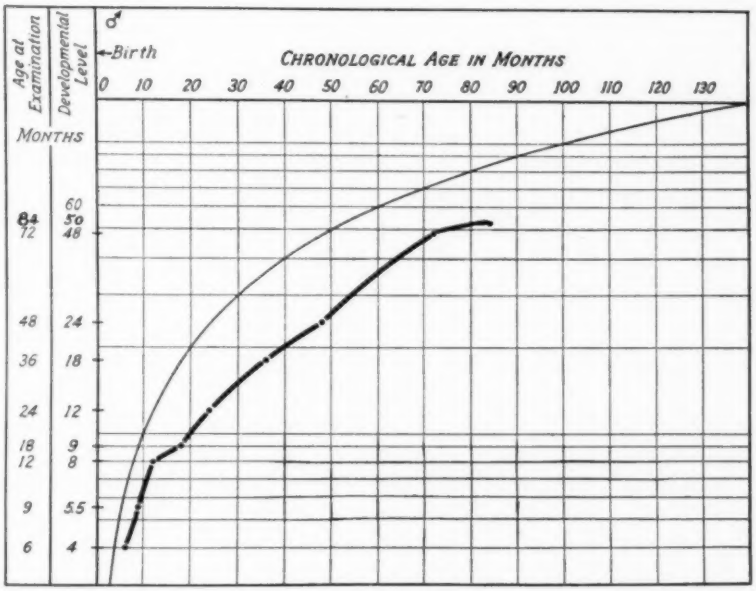

Chart 4 (case 3).—Showing retardation in the mental growth of V. W. from 6 months to 7 years of age, with early physical subnormality.

COMMENT

These three cases are, perhaps, sufficient to illustrate my main thesis, namely, the importance of small amounts of duration in the diagnosis of development in early infancy. The developmental history of the healthy premature infant confirms this thesis in an interesting manner. In him there is a spurious degree of retardation which simulates mental defect; but the false retardation is resolved when his maturity and his age are reckoned from the true base line of conception. Then his mental growth curve takes on a normal conformation. Chronologic age is so consequential that careful clinical discount must be made for the interval of prematurity. For example, a healthy child born two months prematurely may at the age of 6 months present a four months' picture of behavior. If he were a full term infant, this might indicate serious

TABLE 2.—*Relative Measurements of Developmental and Chronologic Age in Case 2*

Examination	1	2	3	4	5	6	7
Developmental age level (in months)	6	8	12	18	24	32	48
Chronologic age (in months)	9	12	18	24	36	45	60

TABLE 3.—*Relative Measurements of Developmental and Chronologic Age in Case 3*

Examination	1	2	3	4	5	6	7	8	9
Developmental age level (in months)	4	5.5	8	9	12	18	24	48	50
Chronologic age (in months)	6	9	12	18	24	26	48	72	84

retardation, even mental deficiency. But since he is a healthy premature child, he is genetically only 4 months old. The developmental status and outlook are actually normal.

There are atypical forms of defect and deviation which cannot be considered here, which place certain limitations on early diagnosis and prediction. Nor can I consider the interesting problem of the differential diagnosis between defect and debility in infancy. In a general way, however, it may be said that even severe physical handicaps, like excessive underweight and malnutrition, do not drastically alter the behavior capacities of the child. In spite of its impressionability, the growth of the nervous system is relatively immune to temporary adversity. There is a stable substrate of neurologic maturation which tends to assert itself in behavior that is characteristic of the child's basic maturity. This stability of the nervous system in the labile complex of growth will increase the possibilities of developmental diagnosis as accurate technic becomes available.

When carefully safeguarded with clinical interpretation, determinations of developmental status, even in early infancy, may be presumed to have a high degree of prognostic import. In a recent study, I summarized the observations in 429 examinations of ninety infants, 80 per cent of whom were examined before they were 18 months of age; twenty-three of the entire group were rated after consecutive examinations as having a developmental quotient of less than 70; most of these children were clinically classified as mentally defective. In all but one instance, the final developmental level was predicted by the observations in the first examination.

Unfortunately, the child does not tend to overcome the subnormality which definitely declares itself in his infancy. This is not surprising, because in the full perspective of both prenatal and postnatal embryology it must be granted that even the infant is far advanced toward his ultimate organization. If he does not show normal characteristics of growth, all is not well with him; for, whether young or old, the human being's capacity for growth is one of the most innate and significant features of his biologic equipment.

In its most characteristic form, mental deficiency does not come through abrupt abbreviation or sharp curtailment of normal growth. It seems rather to express a fundamental, pervasive reduction of growth potency. When the fate of the person rests on such a reduction, a diagnosis of mental defect may be safely made in his early infancy.

Not for scientific reasons alone is it desirable to perfect methods for the early measurement and prediction of developmental status. Even when the defects are insuperable, medicine will place a premium on timely diagnosis as an aid to better control.

OCCLUSION OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY

A CLINICAL STUDY OF FOUR CASES *

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Since Spiller's comprehensive study of the literature and pathologic report of his own two cases,[1] there have appeared at intervals fairly numerous reports emphasizing the definiteness of the clinical syndrome of occlusion of the posterior inferior cerebellar artery. It is important, however, to bear in mind, as pointed out by Spiller and later by Stopford [2] and others, that, in spite of the constancy of the retro-olivary picture, other adjacent vessels cannot always be excluded, especially the vertebral, occlusion of which has been known to present almost precisely the same symptoms. As each of the cases herein recorded displays most of the salient features of the symptom-complex, as well as a few features of individual interest, additional opportunity for the clinical study of medullary function is afforded.

REPORT OF CASES

CASE 1.—*Occlusion of the right posterior inferior cerebellar artery with outstanding oculogyric phenomena: A well marked, spontaneous, transient, conjugate deviation of the eyes to the right; coarse nystagmic movements to the left, horizontal and rotary and not in unison; vertical diplopia. On the right, vegetative signs from involvement of the medullary center of the cervical sympathetic; hemidyssynergia and lateropulsion from involvement of the restiform body; laryngovelopalatine paresis from involvement of the nucleus ambiguus; hemianalgesia and hemithermanesthesia of the face from involvement of the descending root of the fifth cranial nerve. On the left, hemianalgesia and hemithermanesthesia of the neck, trunk and extremities due to involvement of the crossed spinothalamic tract.*

History.—Mrs. E. L., aged 46, was admitted to the Graduate Hospital on May 3, 1928, to the service of Dr. T. H. Weisenburg. The condition began on the previous day with sudden, intense vertigo, in which everything seemed to be whirling about her. Consciousness was not impaired, and she did not fall. A

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* From the Orthopaedic Hospital and Infirmary for Nervous Diseases and the University and Graduate Hospitals of the University of Pennsylvania.

1. Spiller, W. G.: The Symptom-Complex of Occlusion of the Posterior Inferior Cerebellar Artery, J. Nerv. & Ment. Dis. **35**:365, 1908; Tumor of the Gasserian Ganglion, Am. J. M. Sc. **136**:712, 1908.

2. Stopford, S. B.: The Arteries of the Pons and Medulla Oblongata, J. Anat. **50**:131 and 255, 1915-1916; ibid. **51**:250, 1916-1917; The Anatomy of So-Called Deep Sensibility, ibid. **58**:199, 1923.

daughter, having in mind a "stroke," was surprised at the strength displayed in the clasp of the right hand which was held almost constantly within the left and rubbed because of numbness. The eyes were kept closed.

There was nothing of significance in the previous history except that the patient had been undergoing treatment for high blood pressure for a year. She did not use alcohol or tobacco, and said that she had not had venereal disease.

Examination on Admission.—The patient was a fairly intelligent, much overnourished negress, lying quietly with eyes closed but with undisturbed sensorium. She said she preferred the eyes closed, because they were drawn to the right. There was a marked tendency to fall to the right on attempting to sit up or even to raise the head from the bed.

Cranial Nerves: A well marked, spontaneous, conjugate deviation of the eyes to the right was present, with coarse nystagmic movements horizontally to the left. Associated movement to the left was limited in range, but there was no paralysis of the muscles for gaze to the left, the limitation obviously being due to an inability of these muscles to overcome the eyepull to the right. Diplopia was present in all fields due to a lagging of the right superior rectus. To overcome this, either both eyes were kept closed or the left was kept closed or half closed. The pupils were narrow—the right measuring 1.5 mm. in diameter as compared with 2 mm. for the left—and reacted sluggishly to light and in accommodation. Convergence and associated movements of the eyes downward were normal. The fundi showed a high degree of angiosclerosis, more marked on the right. There was no papilledema.

Taste, smell, hearing, vision and swallowing were unaffected, as were movements of the face, tongue and muscles of mastication. The right soft palate was weak, and the right vocal cord paralyzed. Articulation was thick and the quality of the voice was said to be changed, but in what respect could not be ascertained.

Motor System: The muscles of the right upper extremity were distinctly diminished in strength, the hand clasp measuring 65 as compared with 90 on the left. The right lower limb was weak but less so. No disturbance of tone could be distinguished in either arm or leg. Dyssynergia could be demonstrated in the movements of the right arm by the finger-to-nose test and in the right hand by a well marked adiadokokinesis. The right lower limb also showed definite dyssynergia. The left extremities were normally synergic in all movements.

Reflexes: There was no material difference between the reflexes of the two halves of the body, and the plantar reflexes gave a flexor response.

Sensation on the Face: There were occasional severe spasms of painful dysesthesia in the upper right portion of the face. Also in this area sharp, electric-like pains were experienced whenever the left (analgesic) arm was massaged. Both sides responded normally to absorbent cotton, localization, vibration and the compass points, but the right half was insensitive to pinprick, heat and cold. The boundary line of this area of analgesia and thermanesthesia corresponded with the midline in front and in the scalp, passing backward to the region supplied by the cervical plexus and returning in front of the ear and along the lower edge of the right mandible to regain the midline of the chin. Pressure pain was diminished but not lost over this area. The algometer was not employed.

The right cornea was entirely analgesic, and no reflexes could be obtained from it nor from the right conjunctiva. The right nostril and right oral field shared in the impaired sensibility to pinprick, heat and cold. The pharyngeal responses to touch, pressure, pinprick and pressure pain were normal.

anteriorly and from the region supplied by the cervical plexus posteriorly, there

Sensation on the Body: Over the left half, from the region of the clavicle

was defective sensibility to pinprick, pressure pain, heat and cold, which gradually deepened as it included the left arm, trunk and leg. All forms of tactile sensibility were normal everywhere. There was perfect recognition of posture and movement in the limbs in spite of the dyssynergia of the right arm and leg.

Vestibular Examination: This was made on June 6, 1928, and on June 13, 1928, by Dr. Lewis Fisher. By this time, the spontaneous conjugate deviation of the eyes had disappeared, but there remained a well developed, spontaneous nystagmus, chiefly horizontal and rotary, the eyes not moving in unison. The slow movement was to the right and the quick component to the left. Turning tests were not attempted because of the marked tendency of the patient to fall to the right on sitting up. The caloric tests showed normal reactions with the following exceptions: the nystagmus from stimulation of both right and left ears was of prolonged duration; stimulation of the right horizontal canal on the second occasion produced marked conjugate deviation of the eyes to the right but no quick component; stimulation of the left vertical canals on the second occasion produced no response.

On July 3, vertigo and the tendency to fall had disappeared, and the patient was able to sit in a turning chair. Tests by Dr. Fisher at this time showed normal responses except that after turning to both the right and the left, the vertigo thus produced was of short duration, ten to thirteen seconds instead of the normal twenty-five or twenty-six seconds.

Examination of the Sympathetic System: The narrowed right pupil may or may not have been accompanied by other features of the Horner syndrome. Because of the diplopia and the closing of the left eyelids it was impossible to feel sure of any narrowing of the right palpebral fissure or right enophthalmos. Examination by an exophthalmometer was not made. As compared with the left corresponding areas, there was a marked diminution of sweating over the right side of the face and neck and the right shoulder and arm. The area of hypohidrosis on the face was bounded rather sharply by the midline.

Special Examinations: The systolic blood pressure was 228 mm.; the Wassermann test was negative for both the spinal fluid and the blood; there was albumin in the urine.

Subsequent examinations, unfortunately, have not been made because of the removal of the patient to another state. It is known, however, that she reads well and walks about with slight difficulty.

CASE 2.—*Occlusion of the right posterior inferior cerebellar artery with a comparison of the syndrome after thirteen years. On the right, there are still present and unchanged oculosympathetic disturbances, laryngovelopalatine paresis, hemianalgesia and hemithermanesthesia of the face and painful dysesthesias about the eyeball. On the left, there remains in the neck, trunk and extremities impaired response to pinprick, pressure pain and temperature, but to a much less degree than at the onset. Dyssynergia, present in the upper right extremity for some time, is entirely absent as are also disturbances in station and gait.*

History.—Mr. E. L., aged 52, was admitted to the University Hospital on Oct. 9, 1915, under the care of Dr. W. G. Spiller, complaining chiefly of weakness in the right leg. In June of that year, he had suddenly experienced persistent nausea and vomiting and intense vertigo, during which he staggered both to the right and to the left. On the third day, there were difficulty in swallowing, hiccup, thickness in speech and an excessive secretion of saliva. Nasal tube-feeding was necessary for three weeks. Shortly after the onset, there developed severe shooting pains in the right side of the face, mostly about the eyeball, which have persisted. The previous history was unimportant. He had no knowledge of ever having contracted syphilis and denied all use of alcohol and tobacco.

Examination on Admission.—Cranial Nerves: The right palpebral fissure was narrow, measuring 9 mm. in diameter as compared with 10 mm. for the left, and the right pupil was one-third narrower than the left. The conjunctiva on the right, especially within the limits of the narrowed palpebral fissure, was bloodshot. Although there was an apparent right enophthalmos, the exophthalmometer showed the same measurements for each bulb, 15.5 mm. Reactions to light and in accommodation and convergence were normal. There was a pronounced horizontal nystagmus when the patient looked to the right. Both fundi showed slight angiosclerosis.

Muscular movements of the face, mastication and tongue were unimpaired, as were the functions of smell, taste and hearing. There was a slight weakness of the right side of the palate, and the right vocal cord showed no perceptible motility. Swallowing was difficult, and the patient said that the throat felt as if it were filled with fluid.

Motor System: The strength and tone of the muscles of all the extremities were normal. Slight adiadokokinesis was present in the right hand, and the right arm showed marked dyssynergia in the finger-to-nose movement. The movements of the other extremities were normal. The gait was slightly unsteady, and, except for a slight tendency to favor the right ankle which had been fractured some years previously, there was no evidence of staggering. It was noted, however, that in bending backward as far as possible the patient did not flex the knees, showing a lack of proper balance in preventing falling.

Reflexes: There was no material difference between the reflexes on the two halves of the body, except that the plantar response was flexion on the right and absent on the left.

Sensation on the Face: The shooting pains in the right side of the face, felt most keenly in the region of the eye, ceased temporarily after three weeks, to be replaced by a feeling of deep numbness as though located in the jaw bone, and a sensation of cold in the oral cavity as though a piece of ice were held there. Objectively, all forms of tactile response were everywhere normal, but the right side, including the mucous membranes, was defective in response to pinprick and changes of temperature. Impairment in response to pinprick was marked and confined to the distribution of the fifth nerve; that to temperature was distinctly greater in the distribution of the first branch than in the lower two. The right cornea and conjunctiva were analgesic, and their reflexes greatly impaired or even lost.

Sensation on the Body: Numbness was felt over the entire left side, especially in the lower extremity, while the left foot felt "ice cold." In the distribution of the cervical nerves, warmth was less acutely felt than on the corresponding right side. Pain, heat and cold sensibilities were lost in the left arm and trunk and impaired in the left leg. In the latter, heat produced "singular feelings" up and down the limb, causing him to squirm away in marked discomfort and to state "it begins to pain . . . it must be warm." Tactile responses were normal everywhere as were those of position and passive motion in all four extremities. Vibration of the tuning fork was not felt in the upper left extremity but was normally sensed in all other portions.

Vestibular Examination:[3] All responses were normal except that on stimulation of the right horizontal canal an impairment of vertigo was found.

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3. Complete vestibular tests of the patient in case 2 will be found in Jones, I. H.: Equilibrium and Vertigo, Philadelphia, J. B. Lippincott Company, 1918, p. 334.

Subsequent Examinations.—These examinations have been made from year to year by Dr. Spiller and his assistants. On Nov. 5, 1928, thirteen years after the onset, the patient walked with the aid of a cane. The gait was shuffling, but there was no tendency to stagger. The oculosympathetic disturbances, the impaired motility of the right side of the palate and the paralysis of the right vocal cord remained unchanged. Swallowing was unaffected.

Motor System: There was no evidence of disturbed strength, tone, synergy or coordination.

Sensation on the Face: After a brief intermission, the severe spasms of pain, lasting from ten to fifteen minutes, were again persistently present in the region of the right eye. The right side remained defective for sensation to pinprick and changes in temperature and also showed a loss of sensation of pain on pressure. The pressure algometer over the forehead, temple, upper maxilla and chin gave readings of from 0.6 to 1.2 Kg. over the left side, and from 1 to 2.1 Kg. over the right side. The pharynx responded normally to touch, pressure, pinprick and pressure pain, and the gag reflexes were unimpaired. The right cornea remained analgesic.

Sensation on the Body: Subjective disturbances were absent, and the objective observations showed changes. The responses to pinprick, temperature and pressure pain were slightly, if any, impaired in the left arm and trunk, and not at all in the left lower extremity. Both testes were equally sensitive to pressure. Vibratory sensation was normal everywhere.

Examination of the Sympathetic System: The Horner syndrome was still present—the right palpebral fissure measured 7 mm. in diameter and the left 10 mm.; the right pupil measured 3 mm. and the left 4.5 mm. No disturbances in sweating have been noted at any time.

Vestibular Examination: This was made on Oct. 30, 1928, by Dr. Lewis Fisher. The only outstanding feature was a definite oblique perversion of the nystagmus from the right horizontal canal.

Case 3.—*Occlusion of the left posterior inferior cerebellar artery examined one year after the onset of the acute symptoms. On the side of the lesion there were evidences of disturbed function of the medullary center of the cervical sympathetic; hypesthesia, analgesia and thermanesthesia of the face; painful facial dysesthesias; dyssynergia of the upper and lower extremity, and evidences of disturbed function of the nucleus ambiguus (difficulty in swallowing). On the opposite side of the body there were dysesthesias of numbness and impairment of pain and temperature sensibilities.*

Mr. I. Z., admitted to the Infirmary for Nervous Diseases on June 25, 1928, to the service of Dr. T. H. Weisenburg, complained chiefly of pain in the left side of the face and numbness and loss of sensibility to heat and cold in the right arm, trunk and leg. The onset in July, 1927, was characterized by vertigo, nausea and inability to swallow. He was so dizzy that he had to be carried, but there was no disturbance of consciousness. On attempting to walk he staggered, mostly to the left. Sitting in an automobile he thought he was rotating to the right and being carried along in a horizontal position with the head to the right of the road. He remembered seeing double shortly after the onset for a period of a few days, the images appearing one above the other. He became generally weak, but more particularly in the legs. Because of the inability to swallow he was fed by rectum for two months. During this time, it was discovered that he had diabetes. He remembered, within the first few days of the illness, a numbness in the left nostril, dull aching soreness in the left side of the face and a needle-like pain over the

left nasolabial fold. Later, these subjective disturbances developed into knifelike, spasmodic pain extending from the left eyeball to the left side of the chin.

From the beginning there had been a dead feeling over the entire right side of the body, and all objects coming into contact with this area produced a sensation of warmth. Very cold water felt like burning heat; very hot water caused a feeling of heat seemingly deeply situated in the bones. Sensation of pain in the right arm, trunk and leg had been lost.

In 1922, the patient had had a right facial paralysis from which he said he recovered within three weeks. Otherwise the previous medical and social history was unimportant. He did not use alcohol and said that he did not have venereal disease.

Examination on Admission.—Cranial Nerves: There was a slight rotary nystagmus on lateral excursion, more marked on looking to the left. The pupils were unequal, the left smaller than the right, but reacted normally to light and in accommodation. There was no diplopia, and all ocular movements were normal in range. The fundi and fields of vision appeared normal. Smell, hearing and vision were normal, but taste was impaired in the left side of the tongue to sweet and salt. Movements of the lower right side of the face were somewhat weak as a result of the facial paralysis of 1922, but other facial movements were normal as were those of the tongue, mastication and soft palate. Swallowing was unaffected. A laryngeal examination was not made, but phonation seemed normal.

Motor System: No difference could be determined in the strength or tone of the muscles of the extremities of either side. The left arm demonstrated hypermetria and slight decomposition in the finger-to-nose movement, impaired check when the flexed forearm was forcibly extended and suddenly released and a dropping of the extremity below the proper level when asked to bring it from a raised position to a horizontal one. The movements of the left lower limb were slightly dyssynergic in the heel-to-knee and heel-to-toe tests. No disturbance of normal synergy was present in the right arm or leg. The gait was slightly unsteady and on a wide base, but there was no tendency to stagger except toward evenings and then only slightly and toward either side.

Sensation on the Face: The knifelike pains over the left side of the face from the eyeball to the chin still persisted, but, whereas they were of about half a minute's duration, they were present at the time of examination for only a few moments. Numbness also persisted and extended to the whole of the left side of the face, while aching pain was experienced only on shaving. Objectively, the right side of the face and head was normal, but the left side, including the mucous membranes of the nose and mouth, showed the following changes: slight impairment in the response to absorbent cotton; lost sensibility to pinprick, diminished sensibility to pressure and pressure pain and inability to recognize high temperatures. The temperature sense to cold was inverted in the left frontal region, ice being called warm; this sense was normal in the lower regions of the face. On the left, the conjunctival and corneal reflexes were absent; on the right, they were normal. The pharynx was normally responsive to pain and touch, and the gag reflexes were present.

Sensation on the Body: The subjective feeling of deadness on the right side persisted. All forms of tactile sensibility were everywhere normal, as were stereognostic, passive position and motion responses in all four extremities. Sensation of prick began gradually to be defective on the right from the level supplied by the upper cervical nerves. While the loss deepened over the right arm, trunk and leg, at no place was it complete. At times the prick would give rise to perverted sensations as of electric shocks, much less bearable, producing a stronger reaction and referable to an area at least 6 inches (15.24 cm.) in

diameter. Over this same area pressure pain and heat sensibilities were likewise defective but not lost, while cold was sensed as heat. The patient would wince and draw away in apparent great discomfort at the application of extreme cold to either side, correctly pronouncing the stimulus cold on the left and incorrectly hot on the right. The intensity of the discomfort reaction was equal.

Vestibular Examination: A vestibular examination was not made.

Examination of the Sympathetic System: The left pupil measured 3 mm. in diameter as compared with 3.5 mm. for the right, and the left palpebral fissure 7 mm. in diameter as compared with 9 mm. for the right. There was an apparent left enophthalmos, but no examination by the exophthalmometer was made. The left side of the face did not perspire, while the right side did. It was noted that the left side of the face felt warmer and was redder, the small skin vessels of the cheek showing distinct dilatation as compared with those of the right.

Special Examinations: The Wassermann test of the spinal fluid was negative; the blood pressure was: systolic, 120; diastolic, 80; albumin and sugar were present in the urine.

CASE 4.—*Occlusion of the left posterior inferior cerebellar artery examined one week after the onset of acute symptoms. On the left there were oculopupillary vegetative disturbances; palatal, velum and vocal cord weakness with difficult swallowing; temperature dysesthesias and pain and temperature sensory loss over the face, and dyssynergia of the upper and lower extremity. In the neck, trunk and extremities of the opposite side disturbances of pain and temperature sensations were present without any subjective phenomena.*

Miss L. H., aged 67, was admitted to the University Hospital on Jan. 25, 1928, to the service of Dr. W. G. Spiller. The general health had been good until the previous week when she experienced nausea, persistent vomiting and inability to swallow either liquids or solids. She did not strangle, although water seemed to roll around in the throat and gag her. Left frontal headache and dizziness, but apparently no staggering, were present at the onset, but have disappeared. There were no disturbance of consciousness, no tinnitus and no diplopia, but she thought vision was somewhat blurred at times. The previous history was without significance. She had no knowledge of ever having contracted syphilis and never used alcohol or tobacco.

Examination on Admission.—Cranial Nerves: The conjunctiva on the left side was somewhat injected, and the pupil measured 2.5 mm. in diameter while that of the right measured 4 mm. The left palpebral fissure was likewise narrower, measuring 10 mm. in diameter as compared to 12 mm. for the right. Apparently there was a left enophthalmos, but the exophthalmometer showed no difference in the globes, each measuring 18 mm. Intra-ocular and extra-ocular movements were normal, and there was no nystagmus or diplopia. Both fundi showed well defined angiosclerosis. Vision, smell, hearing and taste were normal as were facial movements and those for the tongue and mastication. The left palatal velum was impaired in motility, and there was extreme difficulty in swallowing, necessitating nutritive enemas and nasal tube-feeding. Speech was nasal in quality, and the left vocal cord was paralyzed.

Motor System: There was no demonstrable disturbance of muscular strength or tone. The left arm showed marked dysmetria in the finger-to-nose movements, the left hand considerable adiadokokinesis, and the left heel-to-knee movement was dyssynergic. The movements of the right extremities were normally synergic, the lower being limited by an old ankylosis of the knees. The gait was somewhat unsteady and on a wide base, but there was no staggering. Tendon reflexes, excluding the right patellar, were normal and equal on the two sides.

Sensation on the Face: Subjective: Sensations of chilliness were felt over the left side and not over the right. Objective: All forms of tactile sensibility were everywhere unaffected. The area of distribution of the left fifth nerve, including the nasal and the oral mucous membranes, showed a marked decrease in response to pinprick and changes in temperature, while the corresponding right responses were unaffected. The algometer readings were raised over the forehead, temple, upper maxillary and chin regions of the left side, ranging from 1.2 to 1.6 Kg. as compared to from 0.8 to 1 Kg. over the corresponding regions on the right. On the left, the corneal and conjunctival reflexes were decreased and almost absent; on the right, they were normal. The pharynx was normally sensitive to touch, pinprick and pressure pain, and the gag reflexes were present.

Sensation on the Body: No subjective disturbances were noted. Objectively, from the region supplied by the cervical plexus downward to include the arm, trunk and leg, the right side was defective in response to pinprick, pressure pain and temperature. The algometer readings at different points from the arm to the foot ranged from 1.8 to more than 2.4 Kg. on the right and from 1.2 to 1.8 Kg. on the left. Responses to all other forms of sensory stimuli, including passive position and motion and stereognosis in the extremities, were normal throughout.

Examination of the Sympathetic System: The Horner syndrome and an inability to cry were present on the left side.

Vestibular Examination: A vestibular examination was not made.

Special Examination: The systolic blood pressure was 182, the diastolic 100; the Wassermann test of the blood was negative.

LOCATION AND NATURE OF LESIONS

It is apparent that in each of these four cases essential clinical features exist which localize a lesion in the retro-olivary region of the medulla oblongata on one side. It is also apparent in each, from the character of the onset and tendency toward improvement, that the pathologic process is vascular, indicating occlusion of the artery of supply.

Briefly stated, the medullary localizing features comprise: ipsolateral, hemifacial analgesia and thermanesthesia from involvement of the descending spinal root of the fifth cranial nerve; ipsolateral lateropulsion and hemidyssnergia from involvement of the inferior peduncle of the cerebellum; ipsolateral palatoplegia and laryngoplegia from involvement of the nucleus ambiguus; ipsolateral vegetative disturbances from involvement of the medullary center of the cervical sympathetic; contralateral analgesia and thermanesthesia of the neck, arm and leg from involvement of the crossed spinothalamic tract. These symptoms have been repeatedly described by others and need no further discussion here. The problem arises, however, of attempting to identify with a degree of certainty and definiteness the vascular involvement in each instance.

According to Foix, Hillemand and Schalit,[4] the arterial supply of each side of the medulla comes from three sources in the upper half and from two in the lower half. In the upper half are: the paramedian

4. Foix, C.; Hillemand, P., and Schalit, J.: The Lateral Syndrome of the Medulla and the Blood Supply of the Upper Medulla, Rev. neurol. **41**:160, 1925.

vessels, arising from the basilar, vertebral and anterior spinal arteries, for the supply of the median area including the pyramid and the quadrilateral region extending backward to the fourth ventricle; the artery of the lateral medullary recess, arising from the basilar, for the supply of a wedge-shaped area with its base situated externally between the pyramid and the restiform body; and the posterior inferior cerebellar artery, arising from the vertebral, for the supply of the remainder of the lateral medulla. In the lower half are to be found the paramedial vessels for the supply of the central parts as in the upper half, but the entire remaining lateral portion is supplied by the posterior inferior cerebellar artery to the exclusion of the artery of the lateral recess.

Absence of evidence of inclusion of the pyramidal tracts in all four cases excludes the probability of involvement of the vertebral artery, as is well known, and also, according to these authors, of the paramedian arteries. Furthermore, involvement of the artery of the lateral recess is in no instance likely as the medullary picture points to a wider area of softening than would follow occlusion of this vessel. In brief, each of the four groups of symptoms points to an implication of the lateral area of the medulla, beginning in back of, but not including, the pyramid, and extending posteriorly to involve the restiform body and the medullary center of the cervical sympathetic nerve, i.e., the area of supply of the posterior inferior cerebellar artery. Of course, without pathologic corroboration the evidence at hand can be accepted and weighed only on its clinical merits.

In case 1, because of the prominence of the oculogyratory phenomena, there arises the important question as to the possibility of multiple lesions. Can a spontaneous conjugate deviation to the right, coarse, lateral nystagmic movements to the left and a vertical diplopia be accounted for on the basis of a lesion in the upper part of the right medulla, or is it necessary to postulate one or more concomitant lesions elsewhere? The diplopia was found to be due to faulty action of the right superior rectus muscle. Here, of course, is suggested the possibility of a right third nuclear or nerve involvement, but, as no other muscle supplied by that nucleus or nerve was affected, it is difficult to picture a lesion so small as to select only this portion leaving the others intact.

The spontaneous, conjugate deviation to the right might be due to an irritative lesion of the left cortical center or to a paretic lesion of the right cortical center. Convergence showed that both internal recti were equally operative; in fact, it was demonstrable that there was no weakness of any extra-ocular muscle with the exception of the right superior rectus. Obviously, therefore, if a secondary lesion were to be taken into account, it would have to be of an irritative character. Briefly stated, the diplopia and the conjugate deviation, if due to further patho-

logic changes, would require at least two concomitant lesions for a satisfactory explanation, one in the cerebral cortex and the other in the cerebral peduncle.

Throughout the literature there is occasionally found an observation of disturbances of ocular motility independent of any change in the nuclei of the third, fourth or sixth nerves or their infranuclear fibers. Bickel [5] observed a transient vertical diplopia in the course of an obliterative syndrome of the lateral medullary area due to occlusion of the posterior inferior cerebellar artery. It seemed to him inadmissable that the third nucleus could be involved in a lesion of which all the other symptoms were exclusively bulbar. In an attempt to explain the diplopia as a part of the medullary picture, he called attention to the obvious possibility of disturbance of the labyrintho-oculomotor connections which might result from a lesion of Deiters' nucleus or of the tracts leading from this nucleus to the third, fourth and sixth nuclei. He thought, however, that the explanation in his case was to be found in an involvement of the cervical sympathetic center in the medulla.

Stiefler [6] referred to the vestibular apparatus as a central static organ for the regulation of equilibrium, muscle tone and coordination of head and eye movements. Vestibular eye movements, he stated, occur only with a lesion of the vestibular nerve or its labyrinthine extension, except when its intrapontobulbar end-station is greatly affected.

The lateral vestibular nucleus lies almost exactly in the lateral angle of the fourth ventricle, close to the restiform body and, in a thrombotic process of the posterior inferior cerebellar artery, it is not difficult to see how there might be considerable vestibular irritation, considering the large vascular channnels appearing in connection with it. Eagleton,[7] citing Brunner, stated that conditions of this artery give outstanding signs of vestibular involvement.

Paston [8] cited Mackenzie, Bullock, Fischer and others and offered as an explanation of the multiplicity of ocular motility symptoms in encephalitis not an involvement of the nuclei that preside over ocular motion but, rather, an involvement of some center which has the power of controlling or acting in conjunction with these nuclei. Such a center, he said, can be found in the vestibular nucleus which is definitely

5. Bickel, G.: Contribution à l'étude du mécanisme des troubles oculaires consécutif aux lesions du bulbe rachidien, Rev. gén. d'ophth. **37**:101, 1923.

6. Stiefler, George: Vasculare Erkrankungen im Hirnstam und Kleinhirn, Handbuch der Neurologie des Ohres, Berlin, Urban & Schwarzenberg, 1928, p. 594.

7. Eagleton, W. P.: Otologic and Nasal Diseases in Relation to Intracranial Disturbances, Arch. Otolaryng. **6**:60 (July) 1927.

8. Paston, R. I.: Vestibular or Labyrinthine Epidemic Encephalitis, Brain **40**:482, 1926.

concerned with the relay of fibers from the semicircular canals to the oculomotor nuclei by way of the posterior longitudinal bundle. In encephalitis, he continued, the lesions are commoner in this locality than they are in the region of the third, fourth and sixth nuclei, and any interference with the vestibular nucleus or its arcs would disrupt the perfect responses to passing stimuli and result in abnormal ocular motor phenomena. In other words, Paston was inclined to believe that the ocular symptoms in encephalitis are due to interference with the functions of the vestibular nucleus, arcs or nerves.

Muskens [9] indicated that the regular appearance of nystagmus and conjugate deviation of the eyeballs in variously localized lesions in the cerebral axis points unmistakably to the existence of ascending tracts from the vestibular nuclei. He stated that a lesion of these ascending tracts in man causes only a forced position of the eyes or a still less impressive phenomenon, a tendency toward conjugate deviation.

Antoni [10] was of the opinion that there exists a center for lateral gaze rotation near the nucleus abducens transmitting the cortical impulse to the ocular muscles. He recognized the influence of the vestibular impulses on ocular motility and agreed with the view that both the voluntary and reflex vestibular impulses utilize the same pathways to the nuclei of the ocular muscles.

Leidler [11] described a case of syringobulbia with a lateral gaze to the left, which he thought was the result of a derangement of conjugate movements to the right due to a lesion in the region of Deiters' nucleus on the right side. The conjugate deviation, he stated, reminds one of the eye deviation found in rabbits with injury to the region of Deiters' nucleus. This is probably an instance, unsupported by pathologic observations, of a destructive vestibular lesion in the medulla causing paralysis of gaze to the side of the lesion with predominance of antagonists and a consequent conjugate deviation of the eyes to the opposite side.

Having the thought in mind that the vestibular arcs and not the third nucleus might be responsible for the diplopia, Dr. Fisher, on July 3, 1928, after the conjugate deviation of the eyes had disappeared, stimulated the vertical canals in a sagittal direction by turning. He was able to produce normal and equal movements of vertical nystagmus of good amplitude. If that portion of the third nucleus which presides over the movement of the right superior rectus muscle had been impli-

9. Muskens, L. J. J.: The Central Connections of the Vestibular Nuclei with the Corpus Striatum, and their Significance for Ocular Movements and for Locomotion, Brain **45**:454, 1922.

10. Antoni, Nils: The Syndrome of Internuclear Interruption of the Posterior Longitudinal Bundle, abstr., Arch. Neurol. & Psychiat. **17**:670 (May) 1927.

11. Leidler, B.: Ztschr. f. Ohrenh. **76**:201, 1918.

cated, it might have been expected that the slow vestibular upward pull of the right eyeball under stimulation of the ear would show some impairment. If the involvement had been above the third nucleus, the quick movement upward, or cerebral component, might have been expected to show an impairment. It was the opinion of Dr. Fisher that the resulting eye movements were not indicative of a third nuclear lesion.

As a result of the vestibular examinations of Dr. Fisher the following inferences may be drawn: 1. It is obvious that the oculogyratory disturbances could not have occurred from a lesion within the internal ears themselves, since they were shown to be uninvolved by the presence of normal hearing and the prompt responses to stimulation which were elicited. 2. The shortened period of vertigo after turning indicated a lesion in the brain stem. 3. The spontaneous nystagmic movements, horizontal and rotary, but not in unison (those of one eye being horizontal while those of the other were rotary), point strongly to a lesion situated somewhere between the vestibular nucleus and the nuclei for ocular motility. 4. The absence of response to stimulation of the left vertical canals on the second occasion might indicate that pathologic changes were still in progress involving perhaps decussating vestibular fibers. 5. Nystagmic responses to stimulation (except in the one instance) showed the vestibulo-ocular pathways to be open and the ocular musculature normally responsive. 6. Marked conjugate deviation of the eyes to the right on stimulation of the right horizontal canal, shortly after the subsidence of the spontaneous conjugate deviation to the right, might indicate interference with efferent fibers arising from higher centers for the production of the quick recoil.

Two instances were found in the literature of conjugate deviation of the eyes ascribed to a lesion of the vestibular nucleus in occlusion of the posterior inferior cerebellar artery. Descamps and Quercy [12] reported one in which the deviation was slight and Tilney and Riley [13] referred to the other. In both the gaze was directed toward the side of the lesion. In the absence of pathologic support in these and in the present instance, the question as to whether vestibular irritation or additional lesions may be responsible for the conjugate eye deviation can be considered only from clinical evidence and hypothesis.

COMMENT

The syndrome of the lateral area of the medulla oblongata is fairly well known so that no attempt will be made to consider in detail any but a few salient features relative to the immediate four cases. These have

12. Descamps and Quercy: Rev. neurol. **35**:187, 1919.

13. Tilney, F., and Riley, H. A.: The Forms and Functions of the Central Nervous System, ed. 2, New York, Paul B. Hoeber, 1920, p. 350.

to do chiefly with disturbances and dissociations of functions in the medullary centers and pathways.

Ipsolateral disturbances of the voluntary motor functions of the ninth and tenth cranial nerves occurred in all four cases. The patient in case 1 presented palatoplegia and laryngoplegia; the patient in case 2, persistent palatoplegia and laryngoplegia and transient marked dysphagia; there was no record in case 3 of a laryngeal examination, but a definite history existed of inability to swallow for a period of two months; the patient in case 4 showed palatoplegia, laryngoplegia and marked difficulty in deglutition. Such motor disturbances are explained by the implication within the area of softening of the nucleus ambiguus, which extends throughout the greater part of the medulla in the ventrolateral portion of the reticular formation.

Clinical manifestations of the involvement of this center (syndrome of Avellis), associated with crossed pain and temperature disturbances of the body, offer strong anatomic evidence in all four cases to support the diagnosis of involvement of the posterior inferior cerebellar artery rather than of the vertebral artery, a distinction often impossible to make satisfactorily in the absence of pathologic corroboration. Stopford [2] pointed out that the spinothalamic tract is never supplied by the posterior inferior cerebellar artery below the level of the nucleus ambiguus. The dysphagias, although due to unilateral lesions, were rather complete. According to Spiller [1] this is the rule, recovery in this respect being due probably to the compensating powers of the opposite normal nucleus.

The impairment of taste in case 3, on the side of the tongue involved in the disturbance of pain and temperature, may be assigned to implication of the nucleus solitarius.

Sympathetic Dysfunction.—Disturbance of the cervical sympathetic on the side of the lesion occurred as follows: in case 1, oculopupillary phenomena and diminished sweating, both subsiding within a few months; in case 2, a Horner syndrome, still present and unchanged after thirteen years; in case 3, a Horner syndrome, diminished sweating and vasoconstrictor paresis, all present after one year; in case 4, a Horner syndrome and diminished lachrimal secretion of eleven months' duration.

These changes offer clinical evidence of a fairly wide range of function, as well as dissociation of function, at the level of the center in the midmedullary region. It is of interest to note that all of the disturbances were of a paralytic rather than of an irritative nature and inclined to be permanent with the exception of those in case 1.

Vestibular and Cerebellar Symptoms.—Nystagmus, vertigo and lateropulsion, present in whole or in part in all cases, may be attributed to involvement within the medulla of fibers concerned with the vestibular pathways connected with the spinal cord, cerebellum and oculomotor

nuclei. The hemidyssynergia may be attributed to interference with the spinocerebellar fibers in the inferior cerebellar peduncle.

Dysesthesias.—The occurrence of subjective sensory disturbances is in accord with Wilson's [14] statement that they are commonly associated with organic lesions at the level of the medulla owing to irritation of sensory pathways. When objective changes accompany the subjective, as they do here, he believed that the system involved is partly irritated and partly under destruction. Pains in analgesic areas of the face might thus be accounted for by irritation of the cells in the substantia gelatinosa trigemini from the focus of softening in the medulla. But why, in case 1, pressure of the analgesic left arm should cause sharp pain in the analgesic right side of the face seems rather difficult to explain. Wilson's conception of a combined irritative and destructive lesion might suggest irritation of the cells of the descending root of the fifth nerve by contiguity at a point where the crossed spinothalamic tract concerned adjoins it in the formatio reticularis.

Deep Sensibility.—There was preservation of the so-called muscular sense and deep touch in all cases, due, no doubt, to the integrity of the median fillet. Pressure pain, on the other hand, was affected in all four cases. It was defective in the same areas in the trunk and face as was superficial pain, though possibly to a somewhat less degree. This associated involvement of superficial and deep pain indicates similar or identical pathways for both types, not only in the spinothalamic tract but also in the descending root of the fifth nerve. Wylie [15] and others whom he cited believed that such association exists above the peripheral level but that it is capable of disruption in the medulla oblongata, as superficial pain may be grossly affected in the trigeminal area and deep pain only slightly so or even not at all.

Davis [16] believed that all forms of deep sensibility for the face travel cephalad in the facial nerve and not in the fifth. He cited Mills,[17] Spiller,[18] Head [19] and others and concluded, by applying Head's theory of the sensory mechanism in the spinal nerves to the sensory supply.

14. Wilson, S. A. K.: Dysaesthesiae and Their Neural Correlates, Brain **50**:428 (Oct.) 1927.

15. Wylie, W. G.: Unilateral Bulbar Lesion, Probably Syringobulbia, J. Neurol. & Psychiat. **4**:149, 1923-1924.

16. Davis, Loyal: The Deep Sensibility of the Face, Arch. Neurol. & Psychiat. **9**:283 (March) 1923.

17. Mills, C. K.: Sensory Functions Attributed to the Seventh Nerve, J. Nerv. & Ment. Dis. **37**:273, 1910.

18. Spiller, W. G.: Remarks on Central Representation of Sensation, J. Nerv. & Ment. Dis. **42**:399, 1915.

19. Head, H.: Studies in Neurology, London, Oxford Medical Publications 1 and 2, 1920.

of the face, that the fifth nerve carries the afferent epicritic and protopathic impulses, while the seventh nerve carries impulses of deep sensibility (including pressure pain). It is to be remembered that Head,[19] Stopford [20] and others do not by any means deny the existence of deep sensibility fibers in the skin of the body. Most of such fibers, in their opinion, pass along the tendons and join motor nerves, later separating and entering those posterior roots corresponding in level to the anterior roots for the reception of motor fibers. These authors, however, make the qualification that it has not been proved that deep sensibility is transmitted entirely by fibers accompanying motor nerves and that it is not practical to isolate the sensation of the skin from that of the deeper tissues. This same uncertainty as to the peripheral channels of deep sensibility in the body is no doubt just as applicable to the peripheral pathways in the face, so that most of the fibers may pass cephalad within the seventh nerve and some by way of the fifth. Davis, following the citations mentioned, inclined to the view that the cells of origin of such facial fibers exist in the geniculate ganglion and that their internal prolongations pass centrally within the nervus intermedius.

Pain and Temperature Sensibility.—This was impaired in all four cases on the ipsolateral side of the face and contralateral side of the body. In the face there was no apparent inequality of disturbance in the divisional areas of the fifth nerve, excepting that in case 2 impairment for temperature was greater in the ophthalmic division than in the lower two. In case 3 perception of a cold stimulus as hot was noted in the contralateral side of the body and in the upper distribution of the fifth nerve on the ipsolateral side of the face. Perception of heat in these areas was diminished but not lost. The failure to appreciate cold would suggest a loss of conductivity for that sensation due to destruction, while the impaired appreciation of heat and the sensing of cold as heat might indicate partial destruction and partial irritation of the pathways for heat. An identical dissociation of thermal sensation in both the descending root of the fifth nerve and the spinothalamic tract is thus indicated. It is generally held that, although the paths for pain, heat and cold impulses are frequently affected together in the brain stem, they are independent of one another and any one of the three qualities of sensation may be dissociated from the others by disease.

It is interesting to note that in case 2, after thirteen years, there was almost complete return of sensibility to pain and temperature in the affected half of the body, while in the face the loss in pain and temperature appreciation was only slightly if at all changed. Hun [21] noted the

20. Stopford, S. B.: The Function of the Nucleus of the Trigeminal Nerve, J. Anat. **59**:120, 1924-1925.

21. Hun, H. N. Y.: Med. J. & Rec. **65**:513, 581 and 613, 1897.

same occurrence in his case after three years, although the normal tracts for pain and temperature fibers for the side of the body concerned were destroyed in their course through the medulla oblongata. The explanation he gave is that the sensory loss in the face is due to a lesion of the primary sensory neuron, while that in the body is due to a lesion in a secondary sensory neuron. Because of the numerous branching processes of the cells of the posterior horns of the spinal cord it is possible for impulses to pass by indirect channels. On the other hand, impulses traversing the primary sensory neuron have no collateral branches or indirect channels to follow.

Touch.—All forms of tactile sensibility were everywhere unimpaired in all four cases, except that in the third the response to absorbent cotton was slightly reduced in the affected half of the face. Complete tactile preservation over the body indicates that the median fillet was intact. Complete tactile preservation over the face, except in the one instance of slight impairment on the affected side, coincides with the conclusions of Spiller,[18] Gerard,[22] Stopford [20] and others that tactile impulses from the face do not pass with pain and temperature fibers into the descending root of the fifth nerve, but proceed to the chief sensory nucleus in the pons. Windle,[23] however, in an experimental examination of the sensory root of the trigeminal nerve in the mouse and pig, concluded that probably there was a pathway for tactile impulses to the descending as well as to the main sensory nucleus by way of fibers sending bifurcating branches to both. The destruction of the descending branches of these bifurcating tactile fibers, if they exist in man, might explain the occasional tactile disturbances in clinical pictures of facial analgesia and thermanesthesia after lesions of the spinal tract and its nucleus.

SUMMARY AND CONCLUSIONS

As far as it is possible, without the support of pathologic corroboration, the four cases herein recorded portray acute vascular involvements of the lateral area of the medulla oblongata due to occlusion of the posterior inferior cerebellar artery. For this reason, they may warrant recapitulation of the following points of interest emphasizing our knowledge of the vascular supply and the functions of the tracts and nuclei of this area.

1. While the syndrome of the lateral medullary area is definite and easily recognized, the clinical identification of the particular vessel involved is often difficult.

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22. Gerard, M. W.: Afferent Impulses of the Trigeminal Nerve, Arch. Neurol. & Psychiat. **9**:306 (March) 1923.

23. Windle, W. F.: Nonbifurcating Nerve Fibers of the Trigeminal Tract, J. Comp. Neurol. **40**:229 (Feb.) 1926.

2. Occlusion of the vertebral artery and of the artery of the lateral recess may present clinical features that are almost identical with those of occlusion of the posterior inferior cerebellar artery.

3. Implication of the pyramidal tract, or of the median fillet, is indicative of occlusion of the vertebral or anterior spinal arteries or their branches.

4. Symptoms referable to the posterior portion of the lateral medulla, especially to the restiform body, the vestibular nucleus and the medullary center of the cervical sympathetic, strongly favor occlusion of the posterior inferior cerebellar artery. The syndrome of Avellis, associated with crossed analgesia and thermanesthesia of the trunk and extremities, also favors occlusion of this artery.

5. The vegetative disturbances point to a wide range of function as well as possible dissociation of function of the cervical sympathetic at a midmedullary level. They were all of a destructive rather than of an irritative character and were persistent throughout in three cases.

6. Dissociation of sensory function seems just as possible in a lesion of the descending root of the fifth nerve as in one of the spinothalamic tract.

7. Pressure pain sensibility was impaired in all four cases in the ipsolateral side of the face and the contralateral side of the body but to a slighter degree than superficial pain. Identical or similar pathways for both types of pain sensibility are therefore indicated not only in the spinothalamic tract but also in the descending root of the fifth cranial nerve.

8. Subjective sensory phenomena in analgesic areas, persistently present in all cases, illustrate the frequency of combined irritative and destructive lesions in the medulla.

9. There is some clinical and experimental evidence that at least a few of the fibers of touch may pass with those of pain and temperature into the descending root of the fifth cranial nerve.

10. The symptoms most persistently present are those due to involvement of the descending root of the fifth cranial nerve, the nucleus ambiguus and the spinothalamic tract. In case 2 they were all present after thirteen years.

11. Vestibular symptoms of varying degree were present in all instances.

12. Spontaneous, transient, conjugate deviation of the eyes to the side of the lesion, together with nystagmus to the opposite side and a vertical diplopia, was observed in one case, the question arising as to whether such oculogyric manifestations could be attributed to a lesion of the vestibular nucleus and its arcs or to concomitant higher lesions.

PRIMARY PITUITARY TUMORS

THEIR RELATION TO DYSFUNCTION OF THE ANTERIOR LOBE *

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Tumors of the pituitary body are usually recognized by either or both of two general symptoms: those directly due to pressure of the growth on adjacent structures and those due to glandular dysfunction. The commonest pressure phenomena observed are encroachment on the optic chiasm producing visual field cuts, and deformity of the sella turcica from increased tension within. The changes of glandular origin may be due to a disturbance of the anterior or posterior lobes of the pituitary body, of adjoining tissues, or of distant glands of internal secretion. The functions of these various organs and their interrelationship are not definitely established and this fact makes the interpretation of some of the symptoms of primary pituitary disease difficult.

Some rather conclusive experimental work has been done on the functions of the anterior lobe of the pituitary body. For the purpose of this analysis fifty cases of primary pituitary tumors were selected and studied to correlate some of the symptoms observed with the pathologic data and with the activity of the anterior lobe. The diagnoses were all confirmed at operation, and lesions of adjacent structures which produced pituitary disturbances by extension were omitted.

PATHOLOGY

The cases were classified as follows: adenomas, thirty-one, 62 per cent; cysts, seven, 14 per cent; carcinomas, eight, 16 per cent; adamantinoma, one, 2 per cent; psammoma, one, 2 per cent; glioma, one, 2 per cent, and gumma, one, 2 per cent.

If a definite diagnosis had been made in the seven cases of cystic lesions, the percentage of adenomas might have been greater. The lesions in these seven cases were either merely evacuated at operation, or sufficient material was not obtained for a satisfactory diagnosis. The symptoms in at least two cases suggested that the lesion was adenomatous.

Of the thirty-one adenomas, eight had undergone cystic change. The ages at which symptoms were first observed in this group are shown in the table as ranging from 10 to 56 years, with an average of 27

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* Cases from the Clinic of Dr. Charles H. Frazier, University of Pennsylvania Hospital, Philadelphia.

years. Evidences of glandular dysfunction were probably present at earlier ages, but often were not recognized. Adenomas which had undergone cystic change produced symptoms at an earlier age than did solid lesions. The seven cystic lesions with uncertain microscopic changes occurred in children or young adults and were manifested earlier than most other lesions. The percentage of carcinomas of the

Data on Fifty Cases of Primary Pituitary Tumor

Case	Sex	Age *	Lesion	Dysfunction	Disturbances of Growth
1	F	15	Adenoma (cystic)	Amenorrhea	None
2	F	11	Adenoma (cystic)	Amenorrhea	None
3	M	32	Cyst	Loss of libido	Acromegaly
4	F	23	Cyst	Amenorrhea	None
5	F	30	Adamantinoma	None	None
6	M	33	Adenoma (acido)	Loss of libido	Acromegaly
7	M	29	Adenoma (baso)	None	None
8	M	50	Adenoma (cystic)	None	Scanty hair
9	M	41	Adenoma (acido)	None	None
10	M	25	Adenoma (baso)	None	None
11	M	22	Cyst	Depression	Fröhlich type
12	M	19	Cyst	Depression	Fröhlich type
13	M	15	Adenoma	None	Feminine distribution of hair
14	F	25	Adenocarcinoma	Amenorrhea; loss of libido	None
15	M	45	Adenoma	None	Spacing of teeth; enlarged mandible
16	F	26	Adenoma	Amenorrhea	Acromegaly
17	M	37	Adenoma (cystic)	None	Acromegaly
18	M	52	Adenoma	None	None
19	M	34	Adenocarcinoma	Loss of libido	None
20	F	40	Adenocarcinoma	None	None
21	F	21	Adenoma (cystic)	Amenorrhea; loss of libido	Acromegaly
22	M	31	Adenoma	Loss of libido	Acromegaly
23	F	20	Adenoma (cystic)	Amenorrhea	Skin thick and coarse
24	F	10	Adenoma	Depression	Hair scanty and fine
25	M	25	Adenoma	Loss of libido	None
26	F	15	Cyst	Amenorrhea	Slightly acromegalic
27	F	26	Adenoma (calcification)	Amenorrhea	None
28	F	27	Adenoma	Amenorrhea; sterility	Acromegaly
29	F	30	Adenocarcinoma	Amenorrhea	None
30	M	46	Adenocarcinoma	None	None
31	F	38	Adenoma	Amenorrhea	None
32	F	19	Adenoma	Amenorrhea	Acromegaly
33	M	43	Gumma	None	Scanty hair
34	F	49	Adenocarcinoma	None	None
35	F	26	Cyst	Amenorrhea	None
36	M	3	Psammoma		Dwarfism
37	F	16	Adenoma	Amenorrhea	Acromegaly
38	F	16	Adenoma	Amenorrhea	Slightly acromegalic
39	M	46	Adenoma	Loss of libido	Skin thick and coarse
40	M	45	Adenoma	None	None
41	F	38	Adenoma (cystic)	None	None
42	F	18	Carcinoma	None	None
43	M	28	Cyst, posterior lobe	Loss of libido	Soft skin, scanty hair
44	M	24	Adenoma	Loss of libido	Dwarfism
45	F	32	Adenoma	Amenorrhea	Enlargement of hands
46	M	32	Adenoma	Loss of libido	Acromegaly
47	F	26	Carcinoma	Irregular menses	None
48	M	14	Glioma	Depression	Fröhlich type
49	M	22	Adenoma	Loss of libido	Acromegaly
50	M	20	Adenoma (cystic)	None	None

* The ages listed are those of onset of either glandular or pressure symptoms.

pituitary body is higher than has been observed elsewhere;[1] the onset of symptoms occurred at a later age in these cases than in the cases of adenoma. The average age was 33 years; the youngest patient was 18. Six of the eight malignant lesions were found in women. The other lesions were about equally divided between the sexes.

1. Dott, N. M.; Bailey, P., and Cushing, H.: Hypophysial Adenomata, Brit. J. Surg. **13**:314, 1925-1926.

The four cases of adamantinoma, psammoma, glioma and gumma, respectively, possibly should not be included in this series as they are not of pituitary origin. In all, however, the lesions arose in the pituitary fossa and did not show evidence of extension to other regions.

FUNCTION OF THE ANTERIOR LOBE

In summarizing the literature on the pituitary body, Geiling [2] stated: "The presence of growth-stimulating and ovulation-inhibiting hormones in the pars anterior of the pituitary body seems to be established with certainty." Evans and Long [3] showed this by intraperitoneal injection of bovine anterior pituitary extract into rats. They produced a marked overgrowth, both skeletal and visceral, and sexual repression, manifested by delayed sexual maturity and lengthened or inhibited estral cycles. Similar results could not be obtained by the use of other glandular extracts.

CHANGES IN GROWTH

Skeletal abnormalities and certain typical changes in the skin seem to be definitely associated with the anterior lobe of the pituitary body, whereas adiposity, secondary sex characteristics, basal metabolic rate and distribution of fat may be influenced by the posterior lobe and by other glands. In the cases in this series, only the former will be considered. These changes are as follows:

In the thirty-one cases of adenoma, stimulation of growth was noted in fifteen and retardation of growth in one. In the seven cases of cysts, growth stimulation was noted in two and retardation in two. In the eight cases of carcinoma, growth stimulation or retardation was not noted in any case; in the four cases of other lesions, growth stimulation was not noted in any case, and retardation was noted in two.

Few of these patients presented symptoms before growth was obtained, which can account for the small percentage showing retardation of growth. Fröhlich's syndrome was seen in three cases, two of cystic lesions and one of glioma. The two other patients in whom growth was retarded showed dwarfism, one manifesting pituitary adenoma and the other psammoma.

Stimulation of growth was manifested in varying degrees from coarseness of the skin, spacing of the teeth and enlarged hands and feet, to well developed acromegaly. Cases listed as acromegaly were not all typical, but showed sufficient evidences to warrant this classifica-

2. Geiling, E. M. K.: The Pituitary Body, Physiol. Rev. **6**:62, 1926.

3. Evans, H. M., and Long, J. A.: The Effect of the Anterior Lobe Administered Intraperitoneally upon Growth, Maturity and Oestrus Cycles of the Rat, Anat. Rec. **21**:62, 1921. Evans, H. M.: The Function of the Anterior Hypophysis, Harvey Lectures, 1923-1924, pp. 212-235.

tion. In fifteen of seventeen cases showing stimulation of growth, the presence of adenomas was proved; in the other two cases, cystic lesions of undetermined pathology were present. There is a definite association of acromegaly with pituitary adenomas. Bailey and Davidoff [4] believed that "satisfactory evidence does not exist of the presence of acromegaly without an increase in the number of eosinophilic cells of the pars distalis." Giantism seen in cases of adenoma and produced experimentally by extracts of the anterior lobe suggests a similarity of action of the adenoma and normal cells. However, a counterpart to acromegaly has not been obtained in experimental animals. The suggestion has been made but not proved that the secretion from the adenoma cells, although stimulating to growth, is altered in such a way as to produce these changes.

SEXUAL DISTURBANCES

Amenorrhea is frequently one of the first symptoms of pituitary disease and may be associated with both hypoactivity and hyperactivity of the gland. Teel [5] expressed the belief that the amenorrhea of hypoactivity of the pituitary body is due to ovarian atrophy and that of hyperactivity to large amount of abnormal lutein tissue.

In all but one case of adenoma amenorrhea was present. It was present in all three cases of cyst, whereas in only two of the six cases of carcinoma was this symptom present. It did not exist in the case of adamantinoma. As other pressure phenomena were present in some of the cases which did not show amenorrhea and as this was far more prevalent in the cases of adenoma than in other lesions, it would seem that pressure alone was not responsible for this symptom and that there is perhaps an inhibitory hormone.

Dysfunction in males, manifested by loss of libido and potentia, was recorded in about half the cases without marked predominance in any of the various types of lesions.

SUMMARY

In a series of fifty primary tumors of the pituitary body more than 60 per cent were adenomas. Malignant lesions were found in 16 per cent of cases. Growth stimulation, manifested chiefly by acromegaly, was not found in cases other than in those of adenoma. Amenorrhea was present in thirteen of fourteen cases of adenoma and in only two of six cases of malignant lesions.

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4. Bailey, P., and Davidoff, L. M.: Microscopic Structure of the Hypophysis Cerebri in Acromegaly, Am. J. Path. **1**:185, 1925.

5. Teel, H. M.: The Effects of Injecting Anterior Hypophysial Fluid on the Course of Gestation in the Rat; Am. J. Physiol. **79**:170, 1926-1927.

MENTAL AND NEUROLOGIC CHANGES IN PERNICIOUS ANEMIA

REPORT OF A CASE WITH TREATMENT BY THE MINOT-MURPHY DIET *

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Pernicious anemia is often complicated by lesions of the spinal cord, although these are not markedly advanced in the average case. Owing to the fact that these patients are treated ordinarily by the internist or general practitioner, less attention is paid to the neurologic complications than their frequency deserves, and for the same reason it is probable that many cases with changes of the spinal cord are not reported. Thus the number of cases on record is much smaller than actual experience indicates that it should be.

Grinker [1] listed the percentage of patients showing degeneration of the spinal cord (according to different observers) as follows: Dana, 10; Taylor, 12; Bramwell, 2; Nonne, 2; McCrae, 25; Hannenberg, 50; Woltman, 80 and Grinker, 30 (80 cases of paresthesia are included). Hurst [2] estimated the percentage at 70 and Reese and Beigler,[3] at 80. More recent work tends to verify the higher percentages, namely, from 70 to 80 per cent.

Lichtheim [4] (1887) was the first to mention neurologic changes in the disease that is now called pernicious anemia, and later work has shown the close relationship between this disease and combined sclerosis. Cadwalader,[5] Dickey and McKinley,[6] Service and Baumgartner [7] and

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* Submitted for publication, Jan. 19, 1929.

* Read at a meeting of the Philadelphia Neurological Society, Nov. 23, 1928.

1. Grinker, R. R.: Pernicious Anemia, Achylia Gastrica and Combined Cord Degeneration and Their Relationship, Arch. Int. Med. **38**:292 (Sept.) 1926.

2. Hurst, A. F.: Pathogenesis of Subacute Combined Degeneration of Spinal Cord with Special Reference to Its Connection with Addison's (Pernicious) Anemia, Achlorhydria and Intestinal Infection, Brain **48**:218 (June) 1925.

3. Reese, H. H., and Beigler, S. K.: Subacute Combined Degeneration of Spinal Cord and Pernicious Anemia, Am. J. M. Sc. **171**:194 (Feb.) 1926.

4. Lichtheim: Verhandl d. Kong. f. inn. Med., 1887; Neurol. Centralbl. **6**: 235, 1887; quoted from Bubert.

5. Cadwalader, W. B.: Diagnosis of Subacute Sclerosis of Spinal Cord and Severe Anemia, J. Nerv. & Ment. Dis. **44**:424 (Nov.) 1916

6. Dickey, L. B., and McKinley, J. C.: Subacute Combined Degeneration of Spinal Cord Without Pernicious Anemia; Report of Two Cases with Autopsy Findings, Journal-Lancet **45**:331 (July 15) 1925.

7. Service, S. F., and Baumgartner, E. A.: Subacute Combined Degeneration of Spinal Cord or Pernicious Anemia, Clifton M. Bull. **13**:29 (March) 1927.

many others agreed that the neurologic changes are positive manifestations of pernicious anemia. Hurst [8] stated that "the division between pernicious anemia and subacute combined sclerosis is artificial." Cadwalader [9] (1921) emphasized "the importance of recognizing a group of cases in which the classic picture of combined sclerosis of the cord usually associated with severe anemia is presented, but in which the anemia is absent." Thus one might possibly foretell the onset of the anemia in certain cases and thus aid in influencing the course favorably by beginning intensive treatment early.

Hurst [2] believed that achlorhydria is an essential predisposing cause of the anemia. Knott [10] was of the same opinion, and believed that, due to the achlorhydria, bacteria from above pass into the intestine alive, and also ascend from below. Hurst [2] suggested that both a hemolytic toxin and a neurotoxin are formed in the invaded intestine, and that these two toxins cause the different manifestations of the disease in the blood and the spinal cord, respectively.

The clinical manifestations of the changes in the spinal cord in a typical case may be listed as follows: paresthesias of the extremities; loss of deep sensations—bone sensation and the sense of muscular position; pronounced ataxia and spasticity, worse in the lower extremities; the sensations for touch, pain and temperature not markedly affected except late in the disease, and the tendon reflexes exaggerated with a positive Babinski sign. If the disease progresses to a more extensive involvement of the cord tracts and reflex arcs, the spastic stage may change to a flaccid stage (Cadwalader). The mental changes vary from mild depression and irritability to a classic toxic psychosis, characterized by depressive and persecutory delusions, fearful illusions and hallucinations and an unstable emotional status, with dejection, agitation and confusion. The toxic condition does not often produce a delirium that approaches a psychosis that necessitates special care.

The case of one patient, recently resident for five months in the Pennsylvania Hospital for Mental and Nervous Diseases, was marked by both mental and neurologic complications. This case is of especial interest because in spite of the marked degree of these features, recovery or remission occurred in a relatively short time.

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8. Hurst, A. F.: Addison's Anemia and Subacute Combined Degeneration of Spinal Cord, Lancet **1**:1212 (June 6) 1925; Brit. M. J. **1**:93 (Jan. 19) 1924.

9. Cadwalader, W. B.: The Early Appearance of the Symptoms of Combined Sclerosis of the Spinal Cord and the Subsequent Development of Severe Anemia, Penn. M. J. **24**:692, 1920-1921.

10. Knott, F. A.: Addison's (Pernicious) Anemia and Subacute Combined Degeneration of Cord; Rôle of Achlorhydria and Intestinal Infection, Guy's Hosp. Rep. **177**:1 (Jan.) 1927.

REPORT OF CASE

Clinical History.—A. K. A., a woman, aged 46, was admitted to the hospital on Dec. 8, 1927. Her father had been reticent and strict. He had had bilateral cataract for two years before his death, in 1926, at the age of 84. Her mother had died of tuberculosis of the intestines when the patient was about 10. One brother had died when young from tuberculosis. An aunt had mental trouble during the menopause. A nephew was a wanderer. The patient had three children, two sons, aged 18 and 14, and a daughter, aged 16; all three were normal and doing well in high school or college. There is no history concerning the birth and early development of the patient. She had been a healthy, active child. Three pregnancies had gone to full term, and the first had terminated in a delivery by forceps. No miscarriages had occurred. The menopause had occurred well over two years previous to the illness in question. For ten years, the patient had been troubled with an eruption on the feet under the toes; this condition had become worse about a year previous to admission. There had been gastro-intestinal trouble for many years, and it had become decidedly worse in the two years prior to admission. She had had the ordinary diseases of childhood. There had been no long illnesses and no operations. The patient was a graduate of Vassar and had partly worked her way through college. She had taught in a high school for several years and then had gone into business life as a Y. W. C. A. secretary, etc., which she liked better. She had been successful in everything that she had undertaken.

The husband reported that she had been bright, sociable, and had had many friends. She had been absorbed in her work and her interests had been well rounded; she had been practical and energetic; her views tolerant; she had not been oversensitive, suspicious, or resentful; she had been frank, in general, but not in personal matters. She had not been demonstrative and had not wanted children, but was a good mother. She had met her husband when they were both employed in Christian association work. They had been married two years later, when she was 29; married life was said to have been satisfactory, but the husband had taken a vacation alone in France during the patient's illness, and the patient said that they had discontinued sexual relations in the past few years.

For several years, the patient had had gastro-intestinal trouble which gradually became worse, especially in the two years just before admission. For six months prior to admission, she had had diarrhea, vomiting and belching. A physician had said that her tonsils were diseased; however, she did not want an operation. A physician who saw her in October, 1925, reported that her complaint was a tired feeling, and that she talked rapidly with frequent nervous laughs, had a dark skin, a pulse rate of 92 and a blood pressure of 90 systolic and 60 diastolic. The physician saw her again in June, 1926. She then complained of fatigability, weakness in the fingers and a numbness and prickling in both knees. The blood pressure then was 90 systolic and 60 diastolic, and the hemoglobin was 60 per cent. The physician saw her about once a month thereafter. She lost 26 pounds (11.8 Kg.) in eighteen months; she found it difficult to control her gait. All this time she was overtalkative during office visits, emphasizing her family happiness.

During August, 1927, she visited her stepmother in New Jersey, and while there had severe diarrhea, extreme weakness and nervousness. Soon after her return, in September, the husband went to France for a vacation. While he was gone, she felt the weight of the responsibility of the home heavily and was eager for his return. After his return, she told the physician that she felt that her husband had gone to France to see a French girl whom he had met during the World War. She also told of lying awake at night obsessed by the fear that

he would smother her in her sleep. Her gait became increasingly worse, so that she had to use a cane in walking. For three weeks prior to admission, she accused her husband of taking her jewelry. She thought some one was sprinkling something over her own and her children's faces to make them sleep. She once asked her husband if he would let her go away with the milkman. She called to a passing iceman to come and take her, and told her daughter to call a policeman. On November 25, she was admitted to the Delaware County Hospital, with the diagnosis of anemia (possibly pernicious) and posterolateral sclerosis and from there was sent to the Pennsylvania Hospital for Mental and Nervous Diseases.

Physical Examination.—When admitted, the patient was quiet, apprehensive and dejected. She stated that the change of life had affected her legs and that she could not walk. She showed general weakness, stiffness of the muscles, unsteadiness and inability to use the legs and nervousness.

She was poorly nourished and of the asthenic habitus. The skin was dark olive, much darker around the mouth. There were patches of white on the right foot, surrounded by areas of bronzing; the sclera showed no icterus. Pallor showed in the mucous membranes. The pulse rate was 102; the blood pressure was 127 systolic and 80 diastolic. A mass was found in each upper quadrant of the abdomen and was believed to be a kidney. Examination of the nose and throat showed chronic posterior ethmoiditis and diseased tonsils. The heart and lungs were essentially normal. The spleen was not palpable.

Neurologic Examination.—The ocular movements were full. The pupils were normal in size and shape and reacted promptly and fully to light and in accommodation. There was incontinence of urine and feces occasionally.

Motor Functions: There was pronounced weakness of all muscles, most marked in the legs. The right grip was weaker than the left. The legs showed lead-pipe rigidity with adduction, and were spastically flexed at the knees and hips. The patient had to be supported when standing, and the legs crossed when she attempted to step. She tended to fall forward and double up in a heap.

Coordination: A coarse tremor of the head and an intention tremor of the hands were present. Ataxia was seen in the finger-to-nose and the heel-to-knee tests. Adiadokokinesis was noted. Speech was slow and slightly scanning. The handwriting was cramped.

Reflexes: All reflexes were hyperactive, except the abdominal and epigastric reflexes, which could not be elicited. Achilles clonus was absent or at times transient on the left; on the right it varied from transient to permanent. A pseudopatellar clonus was elicited for a few beats on the right. The Babinski sign was positive bilaterally.

Sensation: The sense of touch was diminished over the trunk, arms and thighs, markedly diminished over the legs and lost in the feet. Pain was slightly diminished over the legs and feet. The sense of heat and that of cold were diminished over the legs and feet. The sense of vibration was normal in the shoulders, diminished in the arms, hands and middle of the back and absent in the sacrum, hips, knees and ankles. The sense of position was diminished in the toes.

Vegetative System: The nails were deeply ridged. The muscles, especially of the lower extremities, were atrophic. The fingers showed clubbing.

Cranial Nerves: The cranial nerves were essentially normal, although there was a question of slight deviation of the tongue to the left, of weakness of the motor divisions of the fifth nerves and of sluggishness of the pharyngeal reflex.

Laboratory Observations: The hemoglobin content was from 70 to 76 per cent; the red cells from 3,170,000 to 3,540,000; the white cells from 5,300 to 8,150; the color index from 0.85 to 1. The red cells seemed normal, and no nucleated red cells were found. The urea content of the blood was normal. The sugar content was 70 mg. per hundred cubic centimeters. (On admission, the color index was 1. The platelets were normal in number. The white cell count on repeated examinations was never over 9,000 and was often as low as 5,500.) The Wassermann reaction was negative. The urine, on three occasions, showed a few pus cells. The results of a van den Bergh test were normal. The gastric secretion showed no pepsin and no free hydrochloric acid. The total acidity was 17. No parasites were found in the feces.

Treatment.—During the time that the patient was in the hospital, she was on the Minot-Murphy diet of liver, with one ampule, twice weekly, of iron citrate (administered hypodermically) and one ampule, once a week, of sodium cacodylate (also given hypodermically). She was given massage every afternoon with negative galvanic electrical stimulation, and heliotherapy (general radiation depending on the reaction of the skin). She was also given $\frac{1}{40}$ grain (1.5 mg.) of strychnine sulphate by mouth for incontinence.

Massage and passive motion of the lower extremities were increased gradually, and then active movements were encouraged. The incontinence ceased in about one month.

Mental Status.—The patient was disoriented much of the time. She forgot what she was doing, and her memory showed patchy defects, but there were clear periods. She felt that all conversation referred to her, and that all noises meant that something was going to happen to her. She was suspicious of every one, and feared that dire things were being planned against her; she believed that her husband was married to some one else, that her family was broken up, and that her daughter was a patient in the hospital. She thought that the telephone wires had been spliced, and that people were testing her out. She felt that she was being compromised. Crying spells were frequent, usually on account of depressive delusions. At times, it seem to be difficult for her to find words with which to express herself. Insight came in flashes; then she admitted for a few minutes that her ideas were delusions caused by her illness. She was restless, getting out of bed frequently and falling, and screaming for the nurses. She crawled on the floor, screaming in terror and trying to escape something.

Diagnosis.—The diagnosis was psychosis with somatic disease; pernicious anemia, with posterolateral sclerosis.

Course in Hospital.—There was a gradual, but steady improvement. She was able to be up in a wheel cahir, wheeling herself around, in three weeks, but still suffered from delusions and was confused and apprehensive. The observations in the laboratory and the neurologic observations showed but little improvement. In six weeks, she was mentally clear, but occasionally depressed. In three months, function was gradually returning to the spastic lower extremities. The hemoglobin had risen to 73 per cent (Dare), and the red blood cells to 4,100,000. Improvement continued until the patient was able to take a few steps. She was again ambitious and in excellent spirits. She was discharged on May 10, 1928, after five months and two days of residence in the hospital, recovered mentally and much improved neurologically.

In September, 1928, the patient was reported as doing satisfactorily. The spasticity of the lower extremities was less. By using a cane, she was able to walk about the house fairly well. She showed no psychosis, but still became worried easily over trifles, and often experienced spells of dejection.

COMMENT

Medical Aspect.—A special point of medical interest is the similarity of this case to Addison's disease. This was noted before the studies in the laboratory were complete. In favor of Addison's disease were the history of weakness, tired feeling, low blood pressure, low content of sugar in the blood and gastro-intestinal symptoms. The discoloration of the skin and the anemia were characteristic of both diseases. The symptoms proceeding from the spinal cord were in favor of pernicious anemia. However, Grinker [1] had reported their occurrence in Addison's disease, carcinoma of the bowel, pellagra and arteriosclerosis. Hurst and Bell [11] had mentioned the association of subacute combined sclerosis with cancer, Addison's disease and lymphatic leukemia. Reese and Beigler [3] also had mentioned the similarity of observations in the two conditions. Despite the interesting similarities, in this case the studies in the laboratory and other physical observations established the diagnosis as pernicious anemia.

Neurologic Aspect.—There was improvement, both subjective and objective, in the neurologic symptoms. This is not the rule in such cases; usually the neurologic symptoms do not remit as readily nor as completely as do the other clinical features. Minot and Murphy,[12] in using the diet of liver, found improvement "except for the pronounced disorders due to spinal cord degeneration." Grinker [1] found, in eleven cases, that no remissions of the neurologic symptoms occurred, although there were remissions otherwise, and he also found that the increase or decrease in number of red blood cells is not related to the neurologic changes. Hurst [2] believed that the amount of the neurologic improvement depends on the amount of toxin affecting the nervous tissue which still remains capable of recovery if the toxin is removed. Cohen [13] reported marked systemic improvement from the Minot-Murphy diet, but his patient not only showed no neurologic improvement, but developed a pronounced ataxia and distressing subjective nervous symptoms. On the other hand, Bubert [14] reported a case in which neurologic and mental improvement was marked. He believed that the Minot-Murphy liver diet is of definite value in cases of subacute combined sclerosis. Service and Baumgartner [7] also found improvement in both the anemia and the neurologic symptoms after three months of a diet of

11. Hurst, A. F., and Bell, O. R.: Brain **45**:266 (Oct.) 1922.

12. Minot, G. R., and Murphy, W. P.: Treatment of Pernicious Anemia by a Special Diet, J. A. M. A. **87**:470 (Aug. 14) 1926.

13. Cohen, A. E.: Subacute Combined Sclerosis Progressive During Remission of Pernicious Anemia; Report of Case, J. A. M. A. **90**:1787 (June 2) 1928.

14. Bubert, H. M.: Subacute Combined Sclerosis: Report of a Case Associated with Pernicious Anemia Improved by Minot-Murphy Diet, J. A. M. A. **90**:903 (March 24) 1928.

liver combined with treatment with eosin and ultraviolet light. These case reports are few and contradictory, and no conclusions can be drawn concerning the neurologic value of the diet of liver until further cases have been studied and reported.

Psychiatric Aspect.—Few cases have been reported in which mention is made of psychic disturbances. Recently, Emile-Weil and Cahen [15] reported one case in which the patient showed "a very marked confusional delirious syndrome." Waterfield [16] mentioned a patient who was depressed, irritable and delirious for five days before death. Bubert's patient showed depression, poor memory and irritability. There are many more who show such mental changes as depression and irritability of various degrees. In case reports, this phase of the reaction of the patient is not described as fully as it should be. Perhaps this has helped to give the impression that the prognosis is grave or hopeless if mental symptoms or delirium develop. The patient in the case here reported was seriously ill in this way, but improved remarkably as did the patients of Emile-Weil and Cahen, and of Bubert.

It is always difficult to evaluate the relation of an organic disease and the psychogenic features associated with it. The factors in this case which may be listed as of psychogenic moment are: The death of the father, the realization of dependency on the husband, and the care of a young family; the fears of infidelity of the husband (who went to Europe on a pleasure trip, without consulting the patient much about it) and the changes in the sexual life in the past few years. The organic factors may be summarized as follows: a primary anemia which was very similar to the usual picture of pernicious anemia, the recent menopause and a toxic delirium (?). In this patient then, there appears to be a toxic state, associated with an organic physical and neurologic disease, inducing a mental reaction (clinically a psychosis) which is colored by the psychogenic factors.

The ultimate outcome is still dubious, and a close "follow-up" will be maintained.

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15. Emile-Weil, P., and Cahen, R.: Presse méd. **36**:744 (June 13) 1928.

16. Waterfield, R. L.: Subacute Combined Degeneration of Spinal Cord with Achylia Gastrica Caused by Atrophic Gastritis, Guy's Hosp. Rep. **77**:93 (Jan.) 1927.

STUDIES IN CHRONAXIA

I. METHODOLOGY AND NORMAL VARIATIONS, WITH A REPORT ON THIRTY CASES IN CHILDREN *

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This study is the first of a series of investigations which we have undertaken on the possible correlation between fundamental neuromuscular responsiveness and the more complex psychomotor capacities in normal persons. Investigations on the separate aspects of the problem have absorbed the interests of both physiologist and psychologist, but correlations between the two thus far are wanting. The main reason, curiously, does not seem to be the absence of a technic for measuring the more complex processes (reaction time, motor speed, etc.) but the difficulty in obtaining reliable measures of neuromuscular responsiveness. This difficulty has seemingly now been removed by the researches of Lapicque and his pupils which furnish physiology with a constant and characteristic measure of neuromuscular excitability, chronaxia. We accordingly attempted to make use of this method in studying its possible availability in the investigation of our problem, and the present paper purports to give a résumé of our experience with it, together with the results obtained by its use on about thirty children.

Chronaxia, as defined by Lapicque, is an experimental constant of excitability which characterizes muscle-nerve responsiveness to electrical stimulation. It is a time threshold—the time required by a current to produce a liminal muscular contraction, with an intensity twice that necessary for it to produce the same contraction when permitted to continue for an indefinitely long duration. More generally, the chronaxia of a muscle or nerve is its electrical time coefficient of excitability or the minimal duration of electrical (galvanic) stimulation that will suffice to evoke from it a liminal response.

This definition differs from the historical view as regards neuromuscular excitability in two important respects: It asserts (1) that the duration of a current as well as its intensity determines its effectiveness as an exciting stimulus, and (2) that the minimal time required for the

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excitation of a particular muscle or nerve (under certain conditions) is a characteristic of its excitability. Both of these generalizations have proved of tremendous significance.

A detailed discussion of the researches which led to these generalizations is beyond the scope of this paper. But it may be pertinent to recall those which are of special interest.

The historical view as to the effectiveness of the galvanic current as a source of stimulation which continued unchallenged from the time when it was first put forth by DuBois-Reymond (1848-1850) until displaced by the modern researches, was that the excitability of a muscle or nerve was exclusively a function of the variation of intensity of the stimulating current and independent of its duration. This view was based on the fact that investigation had shown that the galvanic current acted as an exciting stimulus only on the make or break of the current, whereas no change was perceived during its continued passage. Hence, argued DuBois-Reymond, it must be the change in the intensity of the current and not its actual duration which produced the excitation. Furthermore, in his own attempts to study the influence of curtailing the time of passage of the current on the excitability of muscle and nerve, he had never succeeded in noting any difference in the effectiveness of the current, however short the duration which he employed. He was, however, not aware of the fact that two factors accounted for his failure to observe this: the first, that he was working with a rapid muscle and nerve (the gastrocnemius muscle and the sciatic nerve of the frog), and the second, that his instrumentation did not permit him to obtain a current of sufficiently short duration. The shortest time which DuBois-Reymond employed was about 0.005 second; it happens that the chronaxia of the frog is in the vicinity of 0.0003 second, so that the duration of his shortest current was still more than ten times too large to begin to play any rôle.

During the years following the enunciation of DuBois-Reymond's generalizations, several investigators reported experiments that clearly contradicted the dicta of DuBois-Reymond, but the "laws," as his generalizations came to be known, continued to be accepted until definitely shown to be false by the Dutch physiologist, Hoorweg. In 1892, Hoorweg published the results of a series of experiments on the electrical excitation of motor points in human subjects in which, by the use of condenser discharges, he was able to show that the voltage and, since he was using an approximately constant resistance, the intensity required for effective stimulation varied inversely as the capacity of the condenser employed. Since the time of discharge of a condenser is a function of its capacity, Hoorweg had thus demonstrated that the duration of the passage of a current is not without significance in the excita-

tion of a muscle, and that, on the contrary, there existed a definite relation between this time of passage and the voltage employed.

Weiss (1901), taking up these experiments and substituting directly measured durations instead of condenser discharges, verified the work of Hoorweg and established what has since become known as the laws of Weiss; namely, that the liminal intensity required for the excitation of a muscle or nerve diminished as the time of passage of a current increased up to a certain minimal point beyond which the increase in its duration had no further perceptible effect. The relation was expressed by him as $i.t = a + b.t$, in which t is the time of passage of the liminal current, i the intensity of the current, and a and b two experimental constants which depend on the particular tissue excited and the conditions of the experiment, a being a quantity (Q) and b an intensity (I).

Lapicque's contributions were essentially threefold: (1) he showed that the generalization of Weiss held (approximately) for all muscle and nerve; (2) that the relation of a to b suggested by Weiss as a possible characteristic of irritability was in effect the only constant which characterized neuromuscular excitability, and (3) he furnished a method by which this characteristic, or chronaxia ("chronaxie"), as he named it, could be reliably determined. These contributions [1] have proved of momentous importance and have opened up a new vista of psychologic research and discovery. We mention here only two of them. The first and the more general one is that the chronaxia of a muscle or nerve is closely related to its functional activity. There is a direct relation between the speed of contraction of a muscle and its chronaxia, as is illustrated by the following examples (given by Lapicque):

TABLE 1.—*The Relation Between the Duration of a Muscular Contraction and Chronaxia*

Muscle	Chronaxia, Seconds	Duration of Contraction
Gastrocnemius (frog)	0.0003	0.15-0.20
Claw of crab	0.0030	5
Stomach of frog	0.0100	15-20

The general rule seems to be: with a short chronaxia, a quick response, and with a long chronaxia, a slow response.

The second contribution is that those factors which alter the physiologic function of a muscle or nerve also alter its chronaxia; for instance, temperature, fatigue, degeneration and the action of drugs. Thus, cooling or fatigue lengthen a muscle's chronaxia; increase of

1. Lapicque, L.: L'excitabilité in fonction du temps, Presses Universitaires de France, 1926.

temperature and veratrine shorten it. When a nerve degenerates, its chronaxia is lengthened; as it regenerates, it tends to return to its normal value, etc.

One thus sees that the chronaxia of muscle and nerve is a significant indicator of their functional activity and is influenced by the same factors which affect the organism as a whole. It is these facts which suggested to us the possibility of correlating the differences of psychomotor capacities in general with variations of chronaxia. There was, however, first the problem of methods to be considered. It is therefore the question of the availability of the technic and its practical application with which, as already mentioned, this paper is concerned.

PROCEDURE

The measurement of chronaxia in human subjects, as indicated by Lapicque and perfected by Bourguignon, is effected by the use of condensers. The procedure involves two essential steps:

1. The first is to obtain a liminal response (contraction) of the muscle examined. This is done in a way similar to the method employed in monopolar exploration. One determines the minimal voltage required with a make of a galvanic current, using the negative pole as a stimulating electrode. This is the rheobase of Lapicque.

2. With a current twice this voltage, the experimenter successively charges and discharges a series of condensers, using the same points of stimulation. The capacity just sufficient to produce a contraction equal to that obtained with a rheobasic voltage gives the chronaxia sought.

We shall now discuss each of the required operations.

(1) *Finding the Motor Point and the Rheobasic Voltage.*—The charts for locating motor and sensory points, as given in the textbooks on anatomy and neurology, are of little assistance because at best they are only approximate. In practice, it is necessary to find them for every subject anew. Their precise localization is often both a difficult and a tedious task. The same difficulty is present when one merely seeks a sensory or motor threshold, but for ordinary electrodiagnosis exact localization is not so essential. To obtain accurate measures of chronaxia it is absolutely necessary. When the passage of a current is of short duration, it has not the same opportunity to diffuse as when the duration is indefinite. It is sufficient to displace the stimulating electrodes only a few millimeters to "lose" the point; then one has to start over again. This is particularly true in the case of young children whose nerve trunks are naturally thinner and hence offer smaller "points" at the surface of the skin. Another difficulty is the elasticity of the skin. If one is not careful one risks having the motor point slip from under. It is absolutely essential that the electrode be applied with uniform pressure and without change of direction. Movements, however small, whether of the examiner or of the subject, are likely to throw the electrode off the motor point.

It sometimes happens, after a rheobase with a particular voltage has been obtained, that repetition of the experiment gives a considerably higher or lower value. Such variations almost invariably turn out to be due to inaccurate locali-

zation of the motor point. Even with a small area (from 5 to 15 sq. mm.) which constitutes the motor point region, there is often an optimal or central point. With children we found that displacement of the electrodes for as little as 1 or 2 mm., or even merely changing the angle at which the electrode was applied, made a difference between a normal and a heightened chronaxia.

This last problem brings us to a final important consideration. In searching for the rheobase it is essential to employ unpolarizable electrodes and, what is more important, to avoid polarization of the tissues by the use of continued unidirectional stimulation. This is achieved by changing the direction of the current, through the use of a commutator, with each alternate excitation.

(2) *The Measurement of Chronaxia by the Use of Condensers.*—The measurement of chronaxia, which is a time dimension, by the use of condensers depends on the fact that the rate of discharge of a condenser is a function of its capacity, and the resistance of the circuit across which it is discharged, being proportional to the product RC. If then the capacity is the variable to be measured, the resistance must be kept constant. It happens, however, that the resistance of a tissue, and particularly that of the body, varies from subject to subject and with the conditions of the experiment (temperature, size of electrodes and other factors). Hence, a method must first be found to eliminate or to compensate for these causes of variability. This is achieved by introducing in series and in parallel, a large fixed resistance, such that the subject's individual variation becomes negligible with regard to the total resistance of the circuit. The resistance (R) of the circuit being approximately constant, the chronaxia-time (t) may be calculated as follows: $t = RC \times K$, when C is the capacity used to give the liminal contraction with a rheobasic voltage, and K is the experimental constant, obtained by comparing the condenser discharge with direct rheotomic measurements. This can be done once for all, and was found by Professor Lapicque to be 0.37. The formula then becomes $t = RC \times 0.37$. R being known, t may be calculated by multiplying C by a constant ratio. Thus in Bourguignon's circuit for the measurement of chronaxia in man (which we employed in our investigation), R, the mean reduced resistance, is 11,000 ohms; whence, $t = Ct \times 11{,}000 \times 0.37$, and, expressing Ct in microfarads, $t = 11{,}000 \times 0.000001$ (mf) $\times 0.37 = 0.004$. That is to say, when the resistance of the circuit is 11,000 ohms, $t = Ct \times 0.004$, when t = chronaxia in seconds and Ct, capacities in microfarads.[2]

(3) *Normal Variations in Chronaxia.*—The fundamental contribution of Lapicque to the problem of neuromuscular excitability of a muscle or nerve[3] was a constant which characterized its excitability and was correlated with its functional activity. Different muscles in the same organism and the same muscles in different organisms have their characteristic chronaxias. Bourguignon verified this for the human body and has been able to show by extensive investigation of almost the entire skeletal system that the chronaxias of these muscles classify them into various groups according to their synergetic function. Table 1 gives the figures for the chronaxia of the arm, forearm and hand, given by Bourguignon.

Two facts thus stand out: 1. It is not the anatomic distribution of a muscle or nerve that determines its chronaxia but its synergistic function. 2. There is

2. Bourguignon, G.: La chronaxie chez l'homme, Paris, Masson & Cie, 1923, p. 79. When R is a different value, Ct will have to be multiplied, of course, by another number. The term Ct, designating the minimal capacity needed to obtain a threshold contraction, when seeking a chronaxia, was introduced by Bourguignon.

3. A muscle and its nerve have the same chronaxia.

a definite line of demarcation between the chronaxias of different groups of muscles.

There is yet a third fact, the one which inspired our researches. If one examines the values for chronaxia in each of the groups, one notices that they are not single (mean) figures but represent a range. Thus the chronaxia of the biceps and deltoid muscles is not 0.10 or 0.12 sigma, but from 0.08 to 0.16 sigma; that of the extensor communis digitorum is not 0.40 sigma or 0.60 sigma, etc., but from 0.44 to 0.72 sigma, and so on. The question is whether this range is due to experimental sources of error or whether it represents individual differences. Unfortunately, Bourguignon does not take up this last point. There is some external evidence, however, that the variation is due to individual differences. The range of variability is the same as one finds in the measurement of most biologic and psychologic traits, namely, as 1 : 2.[4] Thus, omitting the extreme

TABLE 2.—*Chronaxias of the Muscles of the Arm, Forearm and Hand, According to Bourguignon*

Group	Muscle	Chronaxia Sigmas
I	Biceps Deltoid Brachialis anticus Supinator longus Internal head of triceps	0.08–0.16
II	External head of triceps Intermediate portion of triceps	0.16–0.32
III	Pronators Superficial and deep flexors Palmaris brevis and longus All muscles of hand Muscles innervated by the median nerve	0.20–0.36
IV	Supinator brevis Extensor carpi ulnaris All the extensors Abductor longus pollicis	0.44–0.77

measures, the weights of white adults fall approximately within the range of from 100 to 180 pounds, their intelligence quotients roughly between 60 and 130, and so on. Indeed, the ratio seems to hold for such different data as the variation in the size of green peas and the weekly salaries of American stenographers.

Now if one examines the chronaxias in figures reported by Bourguignon, one finds that they fall roughly within this range of variation and one might accordingly assume, on *a priori* grounds, that they represent individual differences.

The question, of course, is whether they do represent such differences. Bourguignon, in presenting his figures, speaks merely of averages (moyens), but it is not clear whether they stand for mean values or average or extreme variations, since no measure of dispersing is given, and the answer necessarily awaits further statistical analysis.

4. The figure is approximate, of course. Often it is more nearly as 2 : 3 and sometimes as high as 2 : 5, depending largely on the true or assumed zero. The generalization is based on unpublished researches by one of us (D. W.) which will appear shortly in a separate article.

In any case, further data and verification seem desirable, and the researches that we have to report are an attempt in this direction.

PERSONAL INVESTIGATIONS

Our efforts consisted in obtaining measures of chronaxia on a series of thirty children between the ages of 7 and 12 years. As a determination of chronaxia takes a considerable time (from ten to fifteen minutes) we limited our preliminary study to three muscles, that is, the biceps, the superficial flexors of the fingers (stimulating over the median nerve) and the extensor communis digitorum. These were chosen because they have distinctly different chronaxias, that is, chronaxias of different orders of magnitude, and because the location of their motor points and the detection of the liminal contraction are much easier than in the case of most other muscles.

Apparatus and Technic.—As regards both method and instrumentation, we followed as closely as possible the technic described and employed by Bourguignon.[5] A description of these has already appeared several times in the literature, but the following outline of our present set-up is given to indicate both its correspondence with that of Bourguignon and any deviations that we may have introduced:

Circuit employed	Same as that used by Bourguignon
Source of current	180 volt storage batteries
Condensers *	0.01 to 1.0 mf. (paper, accuracy 2 per cent)
Resistance of potentiometer	650 ohms
Reduced resistance of circuit	11,000 ohms
Electrodes	Silver and silver chloride, covered with asbestos soaked in normal physiologic solution of sodium chloride
Size	Stimulating (negative) electrode, 1 sq. cm.; indifferent (positive) electrode, 5 × 10 cm.
Area of application †	Stimulating electrode over motor point; indifferent electrode, thigh of right leg

* For the determination of normal chronaxia, condensers of greater capacity are not required.

† Bourguignon attached the indifferent electrode to the subject's chest. In the case of children we found it more convenient to attach it to the place indicated. The reduced size of the indifferent electrode is made necessary by the fact that our subjects were children. Both these deviations from the indicated technic do not, however, influence results in any way.

RESULTS

The following readings were taken: (1) rheobasic voltage, (2) the intensity of the current (milliamperes passing through the subject at rheobasic voltage) and (3) the capacity (Ct) to obtain minimum contraction with the double rheobasic voltage.

Three readings were taken during the course of each examination, making nine chronaxia determinations per subject, or three for each muscle examined. In making our calculations, the median of the three measures taken in each case was used.

5. Bourguignon (footnote 2, chap. 3).

The analyses of our observations are summarized in the following four tables:

TABLE 3.—*Current Intensity of Rheobasis*

Muscle *	Average, Milliamperes	Standard Deviation, Milliamperes	Total Range, Milliamperes
Biceps	1.03	0.37	0.6–1.8
Superficial flexor of fingers	1.07	0.38	0.3–1.4
Extensor communis digitorum	1.56	0.42	0.8–1.3

* The region of stimulation was the motor point of the muscle; in the case of the flexors of the fingers, the median nerve.

TABLE 4.—*Chronaxia in MF (Ct)*

Muscle	Average, Microfarads	Standard Deviation, Microfarads	Total Range, Microfarads
Biceps	3.28	0.69	2–5
Superficial flexor of fingers	6.27	1.30	4–9
Extensor communis digitorum	14.40	2.70	12–21.5

TABLE 5.—*Chronaxia (t) in Thousandths of Seconds (Sigma)*

Muscle	Average, Sigma	Standard Deviation, Sigma	Total Range, Sigma
Biceps	0.131	0.038	0.08–0.20
Superficial flexor of fingers	0.251	0.052	0.16–0.36
Extensor communis digitorum	0.576	0.108	0.48–0.85

TABLE 6.—*The Comparative Range of Chronaxia*

Muscle	Bourgignon's Range, Sigma	Total Range in Our Experiment, Sigma	Per Cent of Our Measures Falling within Bourgignon's Range
Biceps	0.08–0.16	0.08–0.20	90
Superficial flexor of fingers	0.20–0.36	0.16–0.36	93
Extensor communis digitorum	0.44–0.76	0.40–0.85	89.3

Table 3 gives the variation in intensity of current needed to evoke a liminal contraction at the rheobasic voltage. This corresponds to the familiar cathodal closure contraction used in electrodiagnosis. The intensity required varied roughly from 0.5 to 2 milliamperes, being practically identical for the superficial flexors and the biceps muscles, and somewhat larger for the stimulation of the extensor communis digitorum. The identity in figures for the biceps and the median again illustrates well how mere intensity of current fails to reveal any fundamental difference in muscular excitability. The higher milliamperage required for stimulating the extensor muscle is not due to the greater intensity of current needed for effective stimulation per se, but to the fact that in the case of the extensor muscle there is a greater diffusion of the current

before it reaches the motor point, owing to the somewhat hidden position of the nerve. In any case, it should be noted that there is much overlapping in the total ranges of amperage for the three muscles.

Tables 4 and 5 give the values for the chronaxia of the three muscles tested in terms of microfarads and sigmas, respectively. Table 5 is derived from table 4 by multiplying the values for chronaxia in microfarads by 0.004 second. The figures show: 1. There is a close correspondence with the values given by Bourguignon. The chronaxias of muscles and nerves in children, within the ages specified, are thus the same as those in adults. 2. The total range of variance in normal chronaxia which we found is somewhat larger than that indicated by Bourguignon (see table 6) even for our median values. But here two facts must be considered: In the first place, we do not know whether Bourguignon's extreme figures represent total range or not. In the second place, it is possible, and indeed probable, that owing to our limited experience with the method, our figures are not so accurate as they might be. While the chronaxia technic as such is simple, considerable practice is required for making reliable determinations. Nevertheless, comparison of our total range with the extreme figures given by Bourguignon shows that for the biceps 90 per cent of our (median) measures, 93 per cent in the case of the superficial flexors, and 90 per cent in the case of the extensor communis digitorum, fall within the range given by Bourguignon.

In addition to these observations we noted, as did Bourguignon and Strohl, that in the case of the extensor muscle one sometimes finds chronaxias of an entirely different order from those expected. Among the twenty-eight cases, in addition to the chronaxias given in table 5, we encountered chronaxias of from 0.24 to 0.40 sigma, or, roughly, approximately one-half that ordinarily characteristic of the extensor communis digitorum. The explanation for the discrepancy, according to Bourguignon, is that the extensors have in reality two different chronaxias corresponding to two different motor points, each correlated with a distinct function of the muscle. Bourguignon observes that whether one hits on the motor point having the higher or lower chronaxia depends largely on whether, when searching for it, one proceeds from below upward or vice versa. In our procedure we were not aware of any systematic method of experimentation, and it would thus seem that the motor point of the higher chronaxia is the more easily located. Like Bourguignon, however, we found that it is always possible to obtain the other by further search.

SUMMARY AND CONCLUSIONS

The normal chronaxia of various muscles in children, aged from 7 to 12 years, is the same as that in the adult.

The extreme range of variation in normal chronaxia for the muscles tested (the biceps, superficial flexors and extensor communis digitorum) is somewhat greater than that given by Bourguignon for adults. It is probable, however, that the discrepancies are due to errors of measurement.

The range of normal chronaxia (the ratio of its extreme limits) is, roughly, as 1 : 2.

The method for measuring chronaxia in men, as perfected by Bourguignon, is simple in both principle and technic, but because of the time, care and practice required for accurate measurements, it appears as yet to be a method for the special technician and research investigator rather than for the general clinician.

While the older clinical determinations of minimum intensity of current required to produce the muscular contraction may suffice for differentiation between normal and gross pathologic conditions, it does not permit the measurement of individual differences in normal neuromuscular excitability. This can be done only through the measurement of chronaxia.

The study of chronaxia opens up a new vista of psychophysiologic research. In view of the fact that the chronaxia of a muscle or nerve is affected by the same factors which influence the functional activity of the organism as a whole (fatigue, etc.), it seems probable that there is a positive correlation between the two. Subsequent studies by us will be devoted to the investigation of possible correlations between chronaxia and psychomotor and neuromuscular tendencies, particularly as manifested in the variability of childhood development.

ABSTRACT OF DISCUSSION

DR. A. B. HIRSH: Ten years ago, while stationed at the Walter Reed Hospital, I read a short paper at an annual meeting of the American Electrotherapeutic Association on some recent advances in muscle-nerve testing; this covered the old formula plus the use of the Lewis Jones condenser and, incidentally, having met at the time Dr. T. A. Williams, just back from Europe, I included material and illustrations which he furnished me on the chronaxia mode of testing. He had intended at that time to include it in a textbook on the treatment of nerve wounds during warfare. At the Walter Reed Hospital we had ample clinical material serving this purpose, but not having the apparatus, we lacked the opportunity of testing chronaxia in our cases. I lost touch with that side of practice for some years, but last summer, while in Europe, I went to the Salpêtrière hoping to meet Bourguignon and have him explain more to me about the technic of chronaxia. He was away on his vacation, however, and his assistants were seen using the method. What interested me particularly was the clinical side of chronaxia. One of the gentlemen present, a New York physician who was studying the method, and who used the French language fluently, acted as interpreter. This brought out the fact that, instead of requiring but three hours for the practical conclusion of one examination, the best part of a day was sometimes devoted to testing one case, and that is what interested me more than

any other phase of the subject. I am not familiar with the physiologists' more recent studies of the method, but the fact that this method, scientific and thorough as it may be, requires so much time, of course, almost precludes its use, viewing it from the clinical point of view.

DR. WECHSLER: I suppose it is because Bourguignon examines so many muscles and nerves that his examination takes so long. During the last few years he has spent a great deal of time gathering material. Actually it takes about fifteen minutes to secure a reliable measurement of chronaxia in a muscle, and much of that time is spent, as in the ordinary clinical test, in finding the motor point. What reduces the time of examination in ordinary electrodiagnosis is that the physician does not have to find it accurately. In the case of chronaxia one does. After a little experience, it took us about thirty minutes per subject to do three complete chronaxias. The values on the chart we made are the medians of nine readings. We took three readings for each muscle, using the middle figure in each case to avoid extreme errors.

DR. JOHN LEVY: I am sure Dr. Wechsler will be good enough to straighten out for me a few points that arose in my mind while listening to his interesting discussion of chronaxia. As I have heretofore understood chronaxia, from my limited reading on the subject, it has seemed to mean a time relationship of nerve tissue. Muscle has acted only as an indicator of this time relationship. Dr. Wechsler sometimes refers to the chronaxia of nerves, sometimes of muscles. Is he not a little vague in that respect?

The next point is the physiologic constant that the speaker sets up for chronaxia. By chopping off both ends of his distribution curve, he is able to establish a 2:1 relationship between the extremes of his chronaxia range. This process seems to me to be artificial. Moreover, it disguises the possibility of a truer relationship. This relationship, or distribution, would more likely follow a normal distribution curve; in other words, it is a continuous series, such as is found in the distribution of intelligence. The fact that the extremes in Dr. Wechsler's range do not peter off to a much higher and lower level, when this 2:1 relationship no longer holds, is probably due to the selectivity of his subjects or the relative grossness of his measuring apparatus.

Has the lecturer any idea as to the possible relationship between chronaxia and intelligence? Some suggestive work has already been done on the relationship between reaction time and intelligence, and chronaxia seems to me to be related to the former. With reference to the possibility of making practical application of chronaxia time, one would be interested in knowing just how constant this time is for the same subject from day to day, and even for the same day. It would surely have to be constant before such application could be made.

I trust Dr. Wechsler will continue to work out these practical points. When this is done, Dr. Pierce Clark may then be able to say that, by means of psychoanalysis, the chronaxia time of the mentally defective patient has been raised eight sigma.

DR. WECHSLER: I am aware of the confusion which indiscriminate and indefinite use of the terms makes, but I have been constrained to repeat the bad usage, because I have never been able to conclude from my reading, whether of textbooks or articles on neurology, what the terms precisely were intended to include. The interchangeable use of muscle, muscle-nerve and nerve, however, does not affect our discussion, because as far as chronaxia is concerned, they are all the same. A muscle and its nerve, as demonstrated by Lapicque, have the same chronaxia.

As to the 2:1 ratio, we used this observation as a source of inspiration only. The thing about it which inspired us was the similarity in the total range of chronaxia with that met with in other biologic and psychologic measurements. This fact indicated that we were dealing with normal variations and not experimental errors. As to dropping 0.5 per cent of the cases, the amount is rather small. We cut off only the extreme portions of the curve, which I think is negligible, when one considers that the remainder includes over 99 per cent of the cases. The importance of the generalization derives not from the fact that the range includes a definite number of cases, but from the fact that it is true in its general aspect.

As regards the correlation between chronaxia and intelligence, I do not know that there are any figures for it. Because of the fertile fields of speculation and investigation which chronaxia has suggested, numerous hypotheses have already been advanced on the basis of it. A book which appeared about two or three years ago attempted to explain all the associative processes on the basis of similarity or identity of neural chronaxia. Association takes place not because of the hypothetic closing up of the dendritic processes, but because a nerve impulse finds its way most readily along paths of nerve processes with the same chronaxias. Irrespective of any theories, however, my personal opinion is that chronaxia will probably not correlate to any degree, if at all, with intelligence, because motor speed enters but little into the intellectual processes.

Dr. Louis S. Aronson: I should like to ask whether, in stating that there is a general constancy for chronaxia, it is relatively or really true in this respect, first, physiologically, and second, pathologically. Physiologically, does it make any difference whether we test out a certain muscle, say the biceps, whether it is a muscle used by brainworkers, or used a great deal for hard work, and second, as regards the age of the subject, would there be a marked difference in the reading of an adult and a child? To come to the second point: Pathologically, have any differences been found in muscles which showed subsequently atrophy or dystrophy, and has there been any experience with muscular atrophies or dystrophies in clinical subjects?

Dr. Wechsler: The first point brought up is one in which we are particularly interested, because it involves the question of individual differences and the factors which may determine them. A variation of several tenths of a sigma may appear negligible when one compares it with the large differences that are met with in the chronaxias of widely different types of musculature (for instance, the gastrocnemius and the muscles of the walls of the stomach in the frog, which have chronaxias of $^{3}/_{10.000}$ second and $^{1}/_{100}$ second, respectively), but may be significant in the case of any particular muscle. Thus the chronaxia of the biceps varies normally only within the range of 0.08 and 0.16 sigma, and it may be that a difference of from 0.04 to 0.06 sigma is sufficient to indicate a fundamental difference in neuromuscular responsiveness. It is differences of this kind, within the normal range, that we hope to study.

As regards the second point, we have no data ourselves, but various French authors, and particularly Bourguignon, give figures on the differences between normal and pathologic chronaxias. The differences are marked in certain myopathies and in the different types of degeneration, the chronaxia of pathologic or degenerated muscles and nerves ranging anywhere from two to one hundred times and more that of the normal.

THE RELATION OF THE CERVICAL SYMPATHETIC TRUNK TO CEREBRAL ANGIOSPASM *

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The opinion that nervous control of the cerebral vasculature was either nonexistent or undemonstrable has long obtained. Although experimental confirmation was lacking, anatomic observations have for years demonstrated the nervous architecture distributed throughout the cerebral vessels. Gulland,[1] although unable to show nerve fibers in his original investigation in conjunction with Bayliss and Hill,[2] was thereafter able to demonstrate their presence.[3] Obersteiner [4] and others [5] were among the earlier investigators employing metallic impregnations in their preparations, and although highly suggestive, these were not viewed as conclusive even from a morphologic point of view. Stöhr,[6] employing a modified Bielschowsky technic, succeeded in demonstrating nervous elements and nerve endings in the pial vascular architecture. It remained, therefore, for Forbes and Wolff [7] to substantiate by accurate photometric methods the observation originally put forth by Donders [8] that faradization of the cervical sympathetic trunk causes constriction of the cerebral arteries. They likewise demonstrated that stimulation of the vagus causes dilatation. This work, having been carried out under direct observation, has been responsible for a revival of the controversy concerning cerebral angiospasm as a fit-producing mechanism.

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* Submitted for publication, Feb. 4, 1929.

* From the Department of Physiology of Oxford University, and the Surgical Clinic of the Peter Bent Brigham Hospital.

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7. Forbes, H. S., and Wolff, H. G.: Cerebral Circulation; Vasomotor Control of Cerebral Vessels, Arch. Neurol. & Psychiat. **19**:1057 (June) 1928.

8. Donders, F. C.: Physiologie des Menschen, ed. 2, Leipsic, Hirtzel and Son, 1859, vol. 1, p. 139; Lancet **5**:521, 1849.

OBSERVATIONS ON THE SPASM OF THE CEREBRAL VESSELS

Of the many phenomena associated with epileptiform seizures, there are several intimately concerned with the cerebral vasculature. Kennedy [9] accurately described a sequence of events ending in a typical grand mal seizure in a patient being operated on under local anesthesia and in whom consciousness and reflex activity were preserved up to the moment of the attack.

"The initial sign of the seizure was a sudden whitening of the cortex which was no sooner remarked on than it was replaced by tremendous venous engorgement with protrusion of the brain beyond the level of the bone defect. This alarming engorgement in which the previously blanched cortex became beet-coloured was coincident with the tonic stage of the attack and the period of general clonic convulsion. The cortex failed to recover its normal appearance during the remaining twenty minutes of exposure although its blood red colour was mitigated in that period. The engorgement was apparently a part of the diffused cyanosis consequent on the general tonic spasm; the initial chalky appearance, however, must have been due to a vascular constriction."

Foerster [10] noted a regular series of events coincident with the onset of unconsciousness, the cortex first becoming pale and sinking away from the encircling rigid integument and subsequently becoming suffused with blood and herniating through the defect as the clonic phase manifests itself.

Of the indirect observations, Hirschfelder [11] noted that the retinal and cerebral arteries contract and dilate synchronously when subjected to epinephrine. Echeverria [12] described the fundus oculi immediately before a fit as being pale with indistinct arteries. Hughlings Jackson [13] noted a pallor of the fundus before the fit, followed by venous suffusion after the onset. Sporadic observations have appeared for years in the literature, and it is of interest to note in Pereira's [14] publication in 1797 the citation of Host's observation that in an epileptic subject in Christiania the left radial pulse invariably disappeared during an attack. At postmortem examination an arterial anomaly was demonstrated, the left arm deriving its blood supply from the vertebral arteries which themselves obtained their supply through the basilar artery from the carotids. The disappearance of the pulse renders it probable that there was an

9. Kennedy, F.: Epilepsy and the Convulsive State, Arch. Neurol. & Psychiat. **9**:567 (May) 1923.

10. Foerster, O.: Deutsche Ztschr. f. Nervenh. **94**:15, 1926; Zentralbl. f. d. ges. Neurol. u. Psychiat. **44**:746, 1926.

11. Hirschfelder, A. D.: J. Pharmacol. & Exper. Therap. **6**:597, 1914.

12. Echeverria, M. G.: On Epilepsy, New York, William Wood & Company, 1870.

13. Jackson, H.: Med. Times & Gaz., Oct. 3, 1863, p. 359.

14. Pereira, J.: The Elements of Materia Medica, ed. 3, London, Longmans, Green & Company, 1854, vol. 2, p. 1797.

arrest of the circulation through the carotids within the cranial cavity during the attack. The behavior of a rapidly exsanguinated animal, and the failure of morbid anatomists to demonstrate satisfactorily on the basis of pathologic tissue changes the mechanism of epilepsy, induced Kussmaul and Tenner [15] to undertake an elaborate study of the vascular elements in the production of epileptiform convulsions. Among their conclusions they stated that the ascending branches of the cervical sympathetic do not appear to be the only channels by which the carotids and their branches receive nerves. Citing the great variability in gross morphology of the sympathetic trunks in the neck, they felt that there was connected with their single successful faradization of the cervical trunk, which resulted in generalized convulsions, considerable anatomic hapstance. They sought to diminish the blood supply somewhat by ligating the contralateral carotid. Cutting and faradization of the cervical sympathetic before ligature of the opposite great vessel resulted in the customarily observed phenomena in the peripheral ear vessels and pupillary dilator mechanism, but on closing the ligature on the opposite carotid they observed that the fundus oculi became extremely pallid, the pupil dilated so widely that the iris could scarcely be seen, the eyeballs protruded, the neck was drawn backward and violent convulsions seized the hind legs, the front legs being obviously securely fastened since the animal was not anesthetized. On removal of the electrodes the convulsions ceased, the fundic pallor receded and the pupil contracted, but the animal remained in a "swooning condition." After some minutes a similar faradization produced an exactly similar seizure, but a third attempt to evoke the phenomena met with failure. They then tied the other carotid, whereupon the animal died without further convulsions. This remains the only direct observation in which stimulation of the cervical sympathetic trunk has been attended by convulsions.

In the light of the establishment of the cervical sympathetic as physiologically capable of diminishing the diameter of pial vessels on an average of 9 per cent when faradized, one is tempted to induce the hypothesis that, robbed of its richly anastomosing collateral circulation of the opposite side, the stimulation of the nervus sympathicus was capable of sufficiently diminishing the cerebral arterial flow to induce a fit-producing anemia. As a closely allied phenomenon, Pike [16] demonstrated that if the cerebral circulation of cats was cut off by tying the internal carotid and vertebral arteries close to the subclavian, unconsciousness was immediately produced and this was shortly followed by asphyxia. If artificial respiration was maintained for a period of from

15. Kussmaul and Tenner: New Sydenham Society, London, 1859.

16. Stewart, G. N.; Guthrie, C. C.; Burnes, R. L., and Pike, F. H.: J. Exper. Med. **8**:289, 1906.

eight to ten minutes and this was followed by a loosening of the ligatures with resumption of cerebral circulation, the animals were thrown into a series of convulsions, epileptoid in character and of a transient incidence varying from a few days to several weeks, preserving a very low threshold, the mechanism being inaugurated at will by jarring, stroking the fur or by acoustic stimulation from noises.

Using a similar technic, Elsberg and Stookey,[17] utilizing a mass ligature, intratracheal anesthesia and artificial respiration, found the optimum time for interruption of the cerebral circulation was eleven minutes, that the convulsions came on in from ten minutes to one hour and could subsequently be evoked for variable periods of time. When an artificial cerebral circulation of Ringer, Locke or saline solution was synchronously begun with the cessation of normal arterial circulation, in a very large series of cases, no fits were observed following resumption of normal blood flow. We shall have occasion to refer to the foregoing as supporting evidence that not only cerebral anemia but cerebral anemia of a very severe grade must occur before fits result either surgically or experimentally.

EXPERIMENTAL WORK

Since the observations are all essentially the same in the six cats used, a single description of the procedure will suffice. Having first anesthetized the cat with ether, the neck was opened in the midline and the carotid sheaths identified. On one side the sympathetic trunk was isolated and a single strand of silk thrown loosely about it. The common carotid artery of the opposite side was then doubly ligated and severed between ties. A stab wound was made through the sternocleidomastoid muscle, and the sympathetic trunk was brought into the subcutaneous space. Here it was sutured into a wide envelope of gutta percha protective. The nerve trunk was then invested with two very delicate copper electrodes spaced from 0.8 to 1.5 cm. apart along its course. The wires and sheets of protective were then led out through and sutured to the midline skin incision. By this means the nerve trunk was well separated from the other physiologically important structures in the neck and current leakage prevented. Dilatation of the pupil was selected as the most satisfactory comparative criterion of sympathetic stimulation. Following recovery from the anesthetic, at times varying from one to twenty-four hours, sympathetic stimulation was carried out in three periods of ten seconds each by means of the usual wet-cell inductorium combination with coil distances beginning at 10 cm. increasing gradually to full strength. One animal was anesthetized by the intraperitoneal injection of iso-amyl-ethyl barbituric acid and subjected to stimulation twenty hours after the implantation of the electrodes.

These preparations all showed extremely active pupillary dilatation. In none of the animals was there any evidence of current leakage; no convulsions, muscle twitchings, increased tonus or loss of consciousness occurred; no pallor of the mucous membranes was noted.

17. Elsberg, C. A., and Stookey, B. P.: Studies on Epilepsy, Arch. Neurol. & Psychiat. **9**:613 (May) 1923.

In a series of ten rabbits the common carotid artery was tied on one side and the sympathetic trunk of the opposite side was secured by a silk tie and divided, distal to the ligature. The nerve was then placed between saline soaked patties and the skin edges were drawn together. At periods varying from thirty minutes to one hour after the stoppage of the anesthesia, and in every case after the animal had returned to full consciousness, stimulation of the nerve trunk was carried out, the coil of the inductorium being placed arbitrarily at 10 cm. and increased to maximum intensity through three periods of ten seconds each which were interrupted by rest periods of several minutes.

Pupillary dilation was more sluggish than in the cat, but was constant throughout in every animal during all three periods of stimulation. There was no loss of consciousness, muscle twitching or rigidity in any of the animals.

COMMENT

The curious roseate hue of the cortex in vivo is due to the color imparted to the opaque tissue medium by the contained blood. This can be readily demonstrated by introducing a dye into the blood stream, the color of the observed cortex changes immediately to that of the dye introduced. The salient fact to direct cortical observation in individuals at the beginning of a fit is the chalk-white appearance of the cortex. In a preparation under ether in which the cortex was exposed, the carotid of the opposite side tied and the sympathetic trunk of the same side stimulated no gross alteration of the pial vessels was observable either to the naked eye or when a 4 × hand lens was employed.

It may be noted that in two animals in the series of Wolff and Forbes no alteration in the established average 9 per cent diminution in caliber of the vessels was noted when ether was employed as a substitute for iso-amyl-ethyl barbituric acid. When an animal is deprived of cerebral circulation for a short time, resumption of circulation is attended by convulsions. If the avascularized head is perfused with saline, recovery is never followed by such convulsions. The direct clinical observations described an obliterative angiospasm producing a startling chalky appearance of the cortex as the immediate precursor of a seizure. Since the establishment of the fact that faradization of the cervical sympathetic trunk contracts the pial vessels the observation of Kussmaul and Tenner has again been subjected to experimental repetition. We are neither able to confirm their observations nor to offer experimental support to the rationale of sympathectomy as a therapeutic measure in the treatment for epilepsy.

CONCLUSIONS

Faradization of the cervical sympathetic trunk in the rabbit and cat on one side in the presence of ligation and division of the common carotid artery of the opposite side is unattended by convulsions.

Clinical and Occasional Notes

ENCEPHALOGRAPHY: THE DEVELOPMENT OF HEMIPLEGIA FOLLOWING ITS USE, WITH REPORT OF A CASE*

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Since the introduction by Dandy, in 1918, of the injection of air into the cerebral ventricles for the purpose of diagnosis and localization of tumors of the brain, this method has been much used by neurosurgeons in all clinical centers and has proved a most valuable adjunct in clearing up a considerable number of obscure and ill defined symptoms of disease of the brain which failed to give the classic tumor syndrome. By this method, early diagnosis before irreparable damage to the brain, or blindness, has occurred and, what is equally important, the surgical approach has been made possible.

It is true that the majority of tumors of the brain are diagnosed clinically, or should be, by meticulous attention to the history and to neurologic signs. A small number, however, elude one no matter how painstaking one is in eliciting the history and conducting the examination. To discuss the reasons for this apparent paradox is obviously not my concern in this communication. Suffice it to say that a well executed and well interpreted injection of air "catches" these elusive or vague cases and raises the diagnostic and localizing percentage to a higher level than was ever attained before.

That the injection of air into the ventricle is not without danger is well known; in inexperienced hands, this injection not uncommonly results in fatalities. Even in the hands of experts, the procedure by no means rarely causes such accidents. The deaths may be attributed to the fact that one has to pierce brain tissue and tear blood vessels; to the sudden emptying of the ventricular fluid and the displacement of the tumor mass with its direct or remote effect on the medullary mass, and last, to the irritating effect of the air itself which may produce a meningitic syndrome.

To obviate these dangers and to remove this important diagnostic measure from the rôle of a major surgical procedure, a number of observers, notably Bingel, Foerster, Wartenberg, Liberson and Carpenter (quoted by Friedman, Snow and Kasanin[1]), have introduced the spinal method of injection of air; it is a simple procedure and yields as much, if not more, information than the method of ventricular injection of Dandy, without the untoward results that sometimes follow the latter procedure. The mortality rate has been negligible and apparently remotely related to the injection of air. Other unpleasant sequelae or accidents have been notably absent.

* Submitted for publication, Feb. 18, 1929.
* From the Department of Neurology, Hospital for Joint Diseases.
* Presented before the New York Neurological Society, March 5, 1929.

1. Friedman, E. D.; Snow, William, and Kasanin, J.: Experiences with Encephalography Via the Lumbar Route, Arch. Neurol. & Psychiat. **19**:762 (May) 1928.

It is my purpose in this report to call attention to the fact that the spinal method may be followed by an unpleasant aftermath, which may be directly charged to the injection.

REPORT OF CASE

History.—M. L., a tailor, aged 45, married, consulted me in December, 1928, for headaches which had been present for one year and which were becoming worse to the extent that he was unable to attend to his occupation. The headaches were intermittent at first but within the past month had become continuous. They were not definitely localized to any part of the cranium, but were diffuse. Vertigo or nausea was not complained of at any time. Visual disturbances were

Fig. 1.—Anteroposterior position visualizing normal "butterfly" appearance of the anterior horns of the lateral ventricles. The third ventricle is in the normal position with possible slight dilatation.

likewise absent. The patient was extremely myopic and had worn glasses for a good many years to correct this defect. A few days prior to coming to the office, he complained of some numbness in the right arm and had difficulty in recalling the names of objects, although there was no difficulty in the power of clear enunciation. A failure of appetite had been noted recently.

The former history of the patient was entirely unimportant. There was no history of acute infections, trauma or indispositions of any sort. He had always considered himself in excellent health, except for a moderate failure in efficiency within recent weeks on account of the headaches.

His parents had died at an advanced age, and there was no history of vascular accidents in the collateral or immediate members of the family.

Examination.—Although the chronologic age was 45, the patient appeared much older than his years. There was no disturbance in gait or station. The pupils were fairly equal, slightly irregular and responded promptly to light and in accommodation. There were no oculomotor palsies. The fundi showed advanced atherosclerotic changes; the edges of the disks showed no pathologic changes except those one notes in high degrees of myopia. Examination of the rest of the cranial nerves showed no deviation from the normal. The skull was not tender to percussion. All deep and superficial reflexes were bilaterally active and equal, and

Fig. 2.—Postero-anterior position visualizing the normal descending and the posterior horns of the lateral ventricles.

no pathologic reflexes were elicited. There were no sensory disturbances and no apraxia. All test objects placed in the hands were accurately named, after some hesitation due to the patient's inability to recall the names of objects. No cerebellar disturbances were noted. The motor system functioned normally.

Mentally, the patient was alert and cooperated in the examination. There was no defect in judgment, insight, orientation or memory.

The systemic examination gave essentially negative results. The heart was not enlarged, and no murmurs were present. The peripheral blood vessels were palpably thickened, and the blood pressure was systolic, 130; diastolic, 80.

In view of the persistence of the headaches, which were apparently increasing, the aphasic phenomena and the paresthesia in the upper extremity, a differential diagnosis between a cerebrovascular disturbance and a neoplasm could not readily be made. The patient was therefore sent to the hospital for more extended observation, which was to include encephalograms.

Course.—Two days later, after a preliminary dose of morphine, ¼ grain (16 mg.), and scopolamine, $\frac{1}{200}$ grain (0.12 mg.), a lumbar puncture was performed. The fluid spurted out under great pressure, and within a brief space of time, 30 cc. was withdrawn. A smaller amount of air was slowly injected. This procedure was continued until 80 cc. of fluid had been withdrawn slowly, although the entire amount could have been removed in a short time on account of the free spurting. Approximately 70 cc. of air was slowly injected. During the

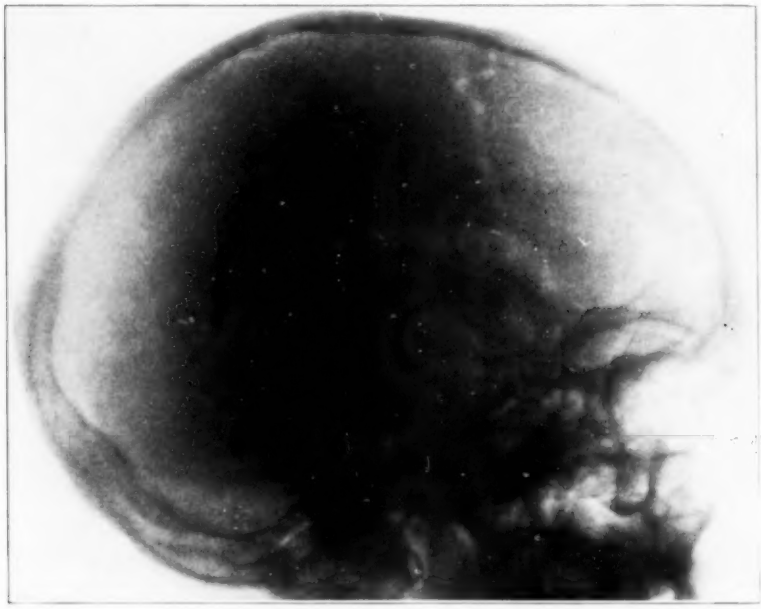

Fig. 3.—Lateral position visualizing the normal lateral ventricles.

procedure the patient complained of some headache, and increasing drowsiness was manifest. No collapse phenomena were noted.

He was immediately taken to the x-ray room, and lateral, anteroposterior and postero-anterior plates of the skull were taken. He was drowsy during and after this procedure and was brought back to the ward. He was seen again two and four hours later, and aside from the continued drowsiness, nothing was noted. When asked how he felt he responded with a reassuring nod of his head.

The following morning, the intern noticed that he was completely aphasic and hemiplegic on the right side. A careful examination corroborated this. The aphasia was absolute; he was unable to utter a single word. The hemiplegia included the leg, arm and face, with exaggeration of the deep reflexes, a Babinski sign and clonic phenomena on the affected side.

The condition remained stationary for about two weeks and was then followed by a gradual regression of the symptoms, and improvement in the motor loss.

At the end of three weeks he was allowed to walk about the wards, which he did in a typical hemiplegic manner. There was little or no improvement in the aphasia up to the time of this report, which was written six weeks after the onset of the disturbance.

A study of the encephalograms revealed entirely negative results as regards distortion, displacement, filling defect or dilatation of the ventricles. The spinal fluid showed 4 cells, no globulin, a negative Wassermann reaction and a flat colloidal gold curve. The Wassermann reaction of the blood was negative. The urine, blood count and blood chemistry showed a condition within normal limits.

COMMENT

The difficulty of proving that the injection of air via the lumbar route, in some unexplained manner, precipitated the hemiplegic syndrome must be obvious to any one who has made use of this simple measure in the detection and localization of brain tumors. The argument can surely be advanced, and not without reason, that on account of the patient's symptoms the inevitable development of a vascular insult was only a matter of time. This is pure conjecture, however, and is of negative rather than of positive value. Cerebrovascular disturbances may exist for months or even years before the final scene in the drama takes place. This is a matter of such common observation that it is unnecessary to dwell on it at length. The development of the hemiplegia in the patient under discussion may have occurred quite early after he came under observation or at some remote period. Predictions in these cases are at best a hazard and are notoriously uncertain. The convincing fact remains that the hemiplegia developed almost immediately after the injection of air.

If one assumes, then, that the injection of air precipitated the hemiplegia, it would obviously be difficult to explain the mechanism responsible for it.

The occurrence of air emboli in the middle cerebral artery is naturally out of the question, in view of the barrier that exists between the blood vessels of the brain and the subarachnoid spaces. The sudden removal of a large amount of ventricular fluid, thus removing a certain amount of support to adjacent inelastic blood vessels, may be considered as an exciting cause, but is hardly tenable in view of the fact that in countless lumbar taps in patients of all ages and conditions no such vascular accidents have been recorded.

Thus, by exclusion, the conviction is forced on one that the air itself in some unexplained manner produced either thrombosis or a rupture of one of the branches of the middle cerebral artery. Possibly an undue amount of distention of the lateral ventricles, caused by the rapid injection of air before the brain mass and blood vessels had time to adjust themselves to the pressure, may have been the responsible factor. That there are flaws in this explanation is evidenced by the fact that the amount of air injected was always less than the amount of fluid withdrawn, and that the total amount of air was less than the total amount of fluid removed.

Perhaps the simplest explanation—unsatisfactory as it seems—is the disturbance caused in the mechanics of the circulation in the blood vessels adjacent to the lateral ventricles. The alternate rapid lowering and elevation of the blood pressure in sclerotic vessels, before proper adjustment takes place, is apparently too much for their weak walls, and a rupture is the result.

SUMMARY

The lesson to be learned from this case may perhaps be summed up briefly as follows:

The preliminary withdrawal of spinal fluid prior to the injection of air should be done gradually, and no spurting of the fluid should be permitted; the air should be injected extremely slowly and deliberately. This obviously is a good rule to follow whatever type of case one is dealing with. It should be followed with particular caution and patience when evidences of arterial disease are present for, as previously noted, the alternate changes in intraventricular pressure occasioned by the removal of fluid and air replacement may, theoretically at least, prove too much for a fragile and inelastic sclerosed blood vessel.

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ABSTRACT OF DISCUSSION

DR. E. D. FRIEDMAN: If one grants Dr. Rosenheck's assumption that the hemiplegia followed in the wake of encephalography, one will have to say that this is the only case on record of such a coincidence. About 200 encephalographies have now been performed under my direction. I have never encountered such a sequel, and I have found no reference to such an occurrence in the literature. In the absence of pathologic studies, it is difficult to say just what the lesion was in this case. It is probable that the patient had a mild cerebral thrombosis to start with and that the process was perhaps aggravated by encephalography. The clinical history, as read, does not justify the diagnosis of hemorrhage into the brain substance. There was no loss of consciousness, and the phenomena usually associated with extensive hemorrhage into the brain were absent. I think that one will have to render a Scotch verdict of "not proved." If there should be further reports of such episodes, it may be necessary to modify the point of view.

When I read my paper before the Section of Neurology and Psychiatry of the New York Academy of Medicine about a year and a half ago, I maintained that there were practically no fatalities in encephalography. Deaths had not occurred in the series of sixty-five cases which formed the basis of the report at that time. Since then the number of encephalographies has grown to nearly 200, and there have been five fatalities. Two of these were immediate — I call them immediate because they occurred within twelve hours of the time the procedure was carried out — and three occurred three or four days thereafter. Three of the cases were tumors of the brain; one was an extensive glioma, deeply seated in the left hemisphere and involving the capsule and the basal ganglia. Following encephalography, the patient developed the syndrome of pseudobulbar palsy and succumbed after three days. The second case was one of endothelioma involving the motor cortex on the right side, high up. Several hours after the procedure was carried out, the patient became stuporous, exhibited slowing of the pulse and vomited profusely. Death occurred within twelve hours, in spite of the withdrawal of spinal fluid and the aspiration of some of the air from the ventricles. The third case was one of an unlocalized tumor with similar symptoms and a fatal issue within twelve hours; no autopsy was obtained. The fourth case was one of progressive subcortical degeneration allied to Schilder's disease (175 cc. of air had been injected). This patient also became stuporous and presented mild meningeal signs. Three days after the encephalography she died. At autopsy, nothing, aside from the degenerative process in the brain, was found to account for the fatal issue. The fifth case was one of amaurotic family idiocy, to which reference has already been made in the first paper on this subject.

I shall therefore have to modify my point of view with regard to the innocuous character of this procedure. In tumors of the brain, one is dealing with a disease in which sudden fatalities are not at all unusual. Dr. Elsberg was kind enough

to tell me of a case in which he had planned to carry out encephalography; before it could be performed, however, the patient suddenly died. If encephalography had been carried out, the death would have been attributed to this procedure. One must therefore be careful in interpreting the significance of the fatal issue in cases of tumor of the brain.

While the procedure of encephalography has a mortality rate, it is not nearly so high as that of ventriculography. According to Grant, a fatal issue occurred in about 8 per cent of 392 cases of ventriculography; in a recent summarization by Heidrich, the average mortality rate was 12 per cent. A recent study by Bingel, covering 895 cases of encephalography, revealed a fatality rate of 0.67 per cent. There is, therefore, considerably less tendency to a fatal issue in encephalography.

One should never attempt to carry out encephalography in cases presenting symptoms of tumor of the posterior fossa. I have unwittingly done so in two instances; fortunately, nothing happened, but I would not deliberately perform encephalography in any case in which the symptoms were strongly suggestive of an expanding lesion in the posterior fossa.

The case report presented by Dr. Rosenheck is interesting from another angle. In my own series of cases there were a number of hemiplegic patients, with and without hypertension. It has been my experience, contrary to his, that most of these patients stand the procedure well. This applies to all persons presenting atrophic disease of the brain — whether it is the result of an old encephalitis, paresis, epilepsy, posttraumatic atrophy or of vascular disease. The coincident phenomena, such as headache and vomiting, seldom occur, and the after-effects are slight. The postencephalographic headache seldom makes its appearance. In this respect, too, the case presented is unique.

I should suggest that the insufflation of air take place slowly, especially at the outset. I never allow 30 cc. to run off freely. At first, I usually withdraw 5 or 10 cc. of fluid and inject a similar quantity of air. I then allow 10 cc. of fluid to escape, and replace it with a little less air. This is repeated until from 80 to 100 cc. of air is injected. In this way one minimizes both the immediate and the after-effects of the procedure. Sometimes, if the fluid escapes freely, I take off 20 cc., but that is the exception and not the rule. When 60 or 70 cc. of air has been injected and one finds that air bubbles appear in the escaping fluid, the injection should be stopped. There is an abnormality in the subarachnoid space and ventricular system of such a person, and the procedure should be discontinued. Ordinarily, one can easily insufflate from 80 to 100 cc. without giving rise to any serious after-effects. In the case of Schilder's disease, 175 cc. of air had been injected. The expansion of this large quantity of air at body temperature may have contributed to the fatal issue. I have also observed that in cases of supratentorial lesions forward and upward, the procedure of encephalography is better borne than in tumors of the temporal lobe.

I shall not spend any time on the discussion of details of the encephalogram, because I shall, in the near future, publish a second paper embodying further experiences with encephalography. I want to say, however, that I believe encephalography has come to stay and that it will ultimately supersede ventriculography, except in cases presenting symptoms highly suggestive of tumor of the posterior fossa and in cases of obstructive internal hydrocephalus. In such instances, the trephine method of Dandy will remain the route of choice.

Dr. J. W. Stephenson: It seems to me that with the development of a new procedure such as this, one should know more about the fatalities, and I will add one more. This condition proved to be an occipital cyst, and the patient was apparently well until 4 a. m. She had some headache and was a little confused,

and she made an attempt to get out of bed; as she did so, she went into a state of coma, and then died with respiratory paralysis. Irrespective of the optimistic view of Dr. Friedman, my own reaction to the injection of air, either by ventriculography or by encephalography, is that it is a dangerous procedure, and I think it should be used only by those who are particularly expert in doing it.

DR. ROSENHECK: I wish to express my gratitude to Dr. Friedman for his lucid explanation of a subject in which all of us are interested. I am not ready to incriminate myself so far as this case is concerned; I simply cited it as an instance of a mishap. Dr. Friedman is correct in his criticism regarding the withdrawal of a large amount of spinal fluid too suddenly. I also agree with him that the air was injected a little too rapidly. Whether the air itself had something to do with the development of the hemiplegia I am not ready to say any more than the points theoretically discussed in my presentation. My conviction is strong, however, that I precipitated the accident. There was nothing to indicate the sudden development of a hemiplegia in this patient. The cerebrovascular phenomena were present, but there was no indication that the patient would develop hemiplegic symptoms at a given time. The fact remains, however, that the paralysis was noted immediately after the injection of air.

I do not intend to convey the impression that I am either condemning or opposing the injection of air via the lumbar route. On the contrary, it is a valuable procedure, and I agree with Dr. Friedman that it has come to stay. It has served me faithfully and well in the cases in which I have applied it, and I hope to make continued use of it.

APPARATUS FOR PNEUMORACHIOCENTESIS*

ARTHUR BERK, M.D., AND PAUL E. TIVNAN, B.S., BOSTON

We present a simple apparatus for the intraspinal and intracisternal injection of air or other gases for the purpose of filling the cerebral ventricles and subarachnoid spaces. This apparatus has the advantage that its component parts are readily available in any laboratory and are easily assembled.

It consists of two 125 cc. separatory funnels with the necessary rubber and glass connections shown in figure 1. Only one of the funnels need be graduated. A volume of 125 cc. has been found sufficient for the usual ventriculography.

The entire apparatus may be sterilized by boiling and filled with sterile water or bichloride solution. Care must be taken that the connections between the lower funnel and the lumbar puncture needle are tight.

The amount of pressure applied to the air under injection can be regulated by varying the distance between the two water levels in the funnels. The rate of flow of the air is easily regulated by the amount that the stopcocks are opened. The amount of air injected is easily measured, as the funnel is graduated in 1 cc. divisions. It has been our custom to inject the air in 10 cc. amounts following removal of 10 cc. of spinal fluid. This process is repeated until the total amount of air is injected.

The needle used is constructed with a two-way stopcock attached, and is the type usually supplied with the Ayer or Fremont-Smith spinal manometer. It has the advantage that the apparatus for the injection of air may be left connected while spinal fluid is being withdrawn or pressure readings are being taken.

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* From the Boston Psychopathic Hospital.

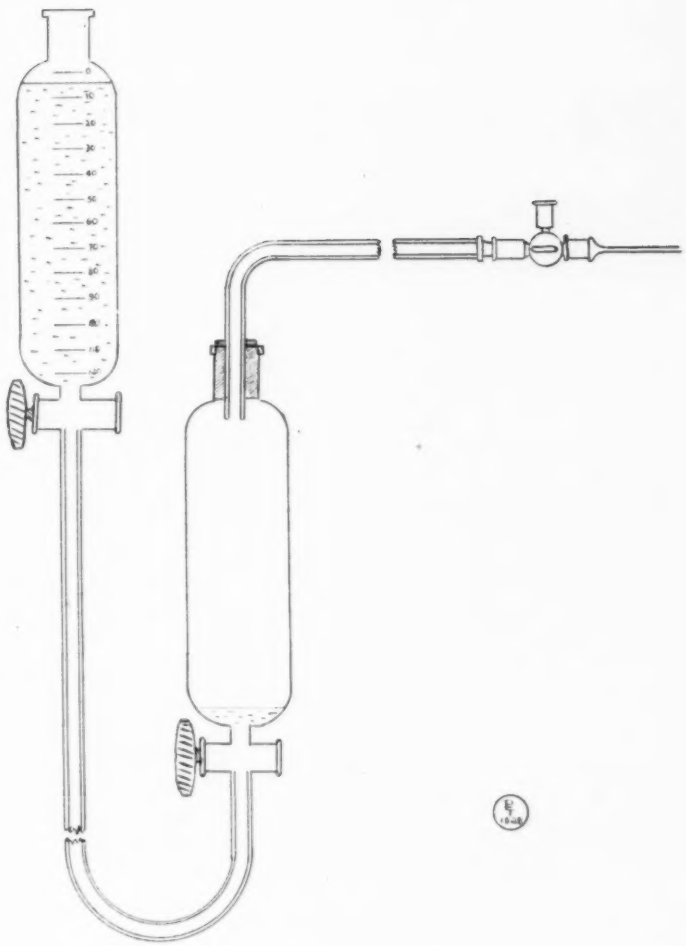

Fig. 1.—A diagrammatic representation of the apparatus.

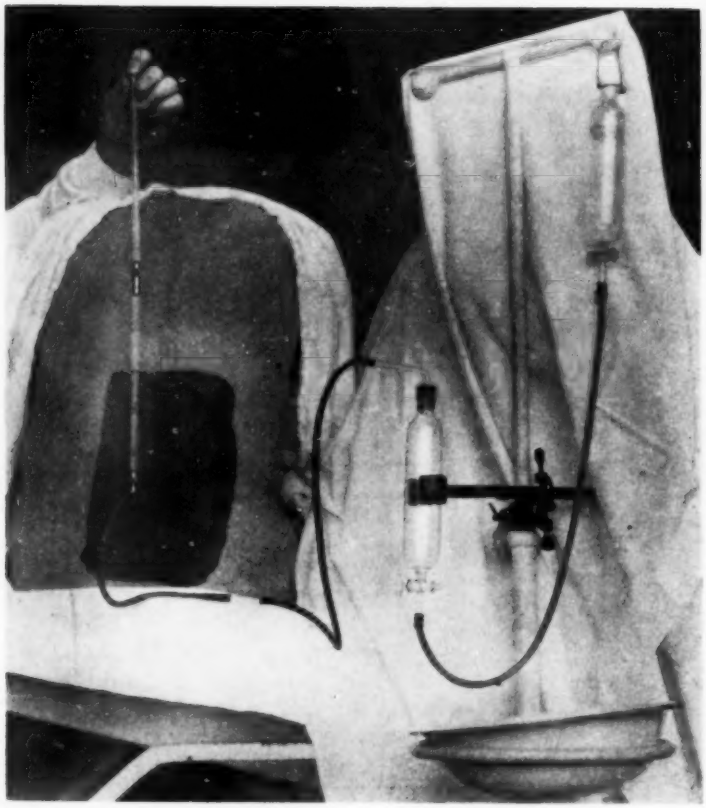

Fig. 2.—A photograph of the apparatus actually in use.

LILLIPUTIAN HALLUCINATIONS

Report of a Case of Hyoscine Poisoning *

ALPHONSE R. VONDERAHE, M.D., CINCINNATI

The atropine series contains a number of closely allied alkaloids, of which the chief are atropine, hyoscyamine and hyoscine or scopolamine. They are found in several plants of the order of *Solanaceae*, and in most cases several of them occur together. The chief plants containing these alkaloids are *Atropa belladonna* (deadly night shade), *Hyoscyamus niger* (henbane) and *Datura stramonium* (thornapple). The leaves of tobacco and potato, also of the order *Solanaceae*, contain minute quantities of these alkaloids. Hyoscine is apparently the least dangerous of the series, for while 0.5 mg. (1/120 grain) is sufficient to cause sleep, over 0.5 Gm. (7½ grains) has been administered to a small cat without causing death.[1]

Few cases of hyoscine poisoning are reported. In the famous Crippen case, in which Dr. Crippen was tried and convicted of poisoning his wife with hyoscine hydrobromide, Wilcox, by chemical and pharmacologic tests, demonstrated the presence of hyoscine hydrobromide in the viscera, amounting in all to 2/5 grain (0.025 Gm.).[2] In Zimmermann's case, a middle-aged man was given 0.007 Gm. (1/10 grain) prior to an operation; unconsciousness developed, artificial respiration was resorted to and the patient recovered in about five hours.[3] Other cases are recorded by Korn,[4] Korff,[5] Blos,[6] Kochmann,[7] Calmann[8] and Nicholson.[9]

In the general literature, diminutive animals and men are frequently mentioned. In the folklore of almost every nation, one finds reference to elves, sprites, dwarfs and fairies. Perhaps the most famous description of minute human beings is that which is found in Swift's "Gulliver's Travels," in which Gulliver makes a voyage

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* Submitted for publication, Nov. 19, 1928.

* From the Department of Anatomy and the Department of Neurology, University of Cincinnati.

1. Cushny, A. R.: Textbook of Pharmacology and Therapeutics, ed. 7, Philadelphia and New York, Lea & Febiger, 1918, p. 331.

2. The Crippen Case, J. A. M. A. **55**:1744 (Nov. 12) 1910.

3. Zimmermann, R.: Ueber einen Fall von Skopolaminvergiftung, München. med. Wchnschr. **59**:423, 1912.

4. Korn, Emil: Ein Fall von Hyoscinvergiftung, Therap. Monatschr. **5**:648, 1891.

5. Korff, B.: Die Narkose des Herrn Dr. Schneiderlin, München. med. Wchnschr. **48**:1169, 1901; Morphin-Scopolamin-Narkose, ibid. **49**:1133, 1902.

6. Blos: Ueber die Schneiderlinsche Skopolamin-Morphin-Narkose, Beitr. z. klin. Chir. **35**:565, 1902.

7. Kochmann, M.: Ueber die therapeutischen Indicationen des Skopolaminum Hydrobromicum, Therap. d. Gegenw. **5**:202, 1903.

8. Calmann, A.: Ein Fall von Skopolaminvergiftung mit gleichzeitigen duodenalen Darmverschluss, Zentralbl. f. Gynäk. **34**:471, 1910.

9. Nicholson, T. G.: Hyoscine Poisoning; Pilocarpine an Efficient Antidote; a Maximum Dose, Lancet **2**:884, 1910.

to Lilliput. It is in recognition of this English classic that the French refer to lilliputian hallucinations.

The medical literature describing hallucinatory experiences involving diminutive human beings is not extensive. The principal student of this type of experience has been Leroy.[10] In the hallucinations that he has described, the subject sees small human beings fantastically and brilliantly costumed and engaged in all sorts of activities. Leroy called attention to the fact that external objects are seen by the subject as of normal size and color. The latter characteristic forms an important point of differentiation from micropsia, in which objective stimuli are all perceived on a diminutive scale. Leroy also pointed out that this type of hallucination is almost invariably agreeable in its emotional content. This pleasant emotional character of the hallucinations may be due, in part, to the fact that the visions are small and harmless, but in greater part it is due to the fact that the subject himself subconsciously believes in a world peopled by small beings who occupy themselves with agreeable pastimes. Leroy[11] observed lilliputian hallucinations in paresis, in a child, aged 7, suffering from measles, and in an old man with senile gangrene. Cases of such hallucinations in other toxic states are also reported: in erysipelas of the face by Salomon;[12] in an old man with cataract by Flournoy;[13] in alcoholism by Colin and Cenac;[14] associated and recurring with each menstrual period by Lelong;[15] and in pulmonary tuberculosis by Alexander.[16]

REPORT OF CASE

The patient, an unmarried woman, aged 50, had not had any occupation other than that of housework. She had always been regarded as extremely nervous, and scrupulous about matters of religion. On the day on which she was first seen, she had consulted a physician about her nervous condition. She took the medicine that he prescribed at 8 p. m. At 11 p. m., she had become extremely restless, was apparently delirious and unable to talk, except in a mumbling fashion.

I saw the patient for the first time at midnight on the same day. At this time, her face was conspicuously flushed. The arms and hands were bright red; the tongue, mouth and skin were dry and parched. The pulse was 100 in rate and

10. Leroy, Raoul: Le syndrôme des hallucinations Lilliputiennes, Encéphale **16**:504 (Nov.) 1921; Le syndrôme des hallucinations Lilliputiennes, Monde méd., Paris, April 18, 1922; quoted by Leroy, J. Ment. Sc. **72**:186 (April) 1926. The Syndrome of Lilliputian Hallucinations, J. Nerv. & Ment. Dis. **56**:325 (Oct.) 1922. Leroy and de Fursac: Les hallucinations Lilliputiennes, Encéphale **15**:189 (March) 1920.

11. Leroy, Raoul: The Affective States in Lilliputian Hallucinations, J. Ment. Sc. **72**:179 (April) 1926.

12. Salomon, J.: Hallucinations Lilliputiennes au cours d'un érysipèle de la face, Soc. clin. med. ment., July, 1920; quoted by Leroy: J. Ment. Sc. **72**:186 (April) 1926.

13. Flournoy, H.: Hallucinations Lilliputiennes atypiques chez un vieillard atteint de cataracte, Encéphale **18**:566 (Nov.) 1923.

14. Colin and Cenac: Alcoholisme et hallucinations Lilliputiennes, Soc. clin. méd. ment., December, 1923; quoted by Leroy: J. Ment. Sc. **72**:186 (April) 1926.

15. Lelong: Hallucinations Lilliputiennes recidivantes à chaque période menstruelle, Soc. med. psychol., March, 1923; quoted by Leroy: J. Ment. Sc. **72**:186 (April) 1926.

16. Alexander, M. C.: Lilliputian Hallucinations, J. Ment. Sc. **72**:187 (April) 1926.

was regular. The pupils were widely dilated and fixed to light. All the deep reflexes were exaggerated. The patient obeyed simple commands, such as to put out the tongue and give her name, but could sustain her attention for only a few seconds. She was extremely restless and constantly attempted to get out of bed. She also mumbled to herself continuously.

The physician stated that he had intended to order powders containing sodium bromide and extract of hyoscyamus. Instead of writing extract of hyoscyamus, however, he wrote hyoscine.

The patient was seen about twenty hours after this episode of delirium. At this time, she was still extremely restless and anxious, but there was a definite subsidence of the toxic symptoms. She recalled, with considerable amusement, some evidence of the delirium of the previous day. The experience that amused her most was the sight of many minute and brightly colored animals that performed queer antics, such as forming into parades and going through fascinating maneuvers, the most striking to the patient being one in which a long and apparently never ending line of horses, giraffes, tigers, elephants and so on proceeded over the top of a mantlepiece to a vase, on which the minute forms climbed and disappeared by toppling into the vase. The patient could not keep from laughing when she recounted her experience.

Eight days after the acute attack, a psychiatric examination of the patient brought out the fact that since the age of 15 she had always been extremely apprehensive about religious matters. She was a Catholic, and never felt sure that her confessions were properly made or that her sins were forgiven. In addition, she was afraid that she would die suddenly of heart disease. A physical examination revealed nothing abnormal except exaggerated reflexes, several carious teeth and a functional tremor of the hands. Examination of the urine gave negative results, and a Wassermann test of the blood showed nothing abnormal. A test of the basal metabolism gave a reading of + 32. The diagnosis made at this time was psychoneurosis, psychasthenic type, and moderate hyperthyroidism.

Leroy considered that lilliputian hallucinations result from the action of various toxins on cortical centers. Certainly, the condition in the majority of cases reported in the literature is associated with a definite toxic factor. Leroy [11] stated that, experimentally, hallucinations of dwarfs can be produced by using alkaloids the physiologic action of which causes brilliance of the eyes, mydriasis, coloration of the face and slackening of the pulse.

There is the "Epyotl," a sacred plant that the natives of Mexico venerate as a divinity, and to which they offer a particular and complicated worship. According to the works of M. Rouhier, the absorption of this intoxicating substance produces a general and pleasing excitement, feelings of lightness, muscular power, euphoria, even exhilaration, then visions of incomparable beauty. Amid these oniric phenomena one very frequently meets with typical colored lilliputian hallucinations. These facts are of important interest in the relationship of the affective state and hallucinations.

There are, however, other theories as to the cause of these phenomena. Alexander [10] stated that while any toxin may justifiably disturb the normal mental balance, this would not explain why these hallucinations take this particular form. He suggested two explanations: 1. The person so affected has an abnormally strong instinct of tenderness, which is kept in its proper place so long as this person maintains a normal mental balance. If, however, the mental balance is overthrown, the instinct of tenderness may predominate over all others, and because such a patient delights in small objects, hallucinations became lilliputian. 2. The

patient suffers from a strong inferiority complex; anything smaller than himself tends to counteract his sense of inferiority and to give him a feeling of strength and power. Lilliputian hallucinations would therefore be those most likely to please him. Flournoy [13] put forward a theory that lilliputian hallucinations represent a reversion to a mental state of childhood, when the patient's interests were chiefly in small things and in small people.

SUMMARY

A case of acute hyoscine poisoning is described. The hyoscine poisoning was associated with the appearance of lilliputian hallucinations. Further evidence of a toxic factor in the production of this phenomenon is adduced.

Abstracts from Current Literature

ARTERIOVENOUS ANEURYSM OF THE BRAIN, AND VENOUS ABNORMALITIES AND ANGIOMAS OF THE BRAIN. WALTER E. DANDY, Arch. Surg. **17**:190 (Aug.) 1928; **17**:715 (Nov.) 1928.

In a series of about 600 verified tumors of the brain, vascular tumors and aneurysms of the brain occurred in about 5 per cent of the cases. Eight of the lesions were arteriovenous, fifteen were referable to the venous and capillary system and seven were arterial. Five of the seven venous abnormalities appeared in the cortical vessels in connection with the sylvian vein. Seven of the eight examples of arteriovenous aneurysm were located in the paracentral region and apparently had an arterial connection with a branch of the middle cerebral artery. Four cases of venous abnormalities in the dura (venous aneurysms as evidenced by a diverticulum of the superior longitudinal sinus) were noted at the top of the motor cortex. The venous abnormalities are commonly accompanied by other deformities in the brain and disturbances in mentality. Signs of intracranial pressure—headaches, choked disk—or of progression of symptoms, all evidences of an expanding lesion, are noticeable by their absence in these cases.

All of the cysts with angiomatous tissue in the wall occurred in the cerebellum or vermis. The symptoms produced were those characteristic of lesions in this area.

The cavernous angiomas were found in the occipital lobe once, in the cerebellum three times, and in the pons and medulla once. At least three striking clinical differences between cavernous angiomas and angiomatous cysts can be shown: 1. Most of the symptoms of cavernous angiomas are focal, and few are of general intracranial pressure. 2. The duration of symptoms from cavernous angiomas is much longer. 3. The symptoms begin at an earlier age.

Cerebral hemorrhage is much more common in cavernous angiomas, venous or arterial abnormalities than in angiomatous cysts. However, except in the cases of prolonged nonprogressive jacksonian epilepsy, in which a local lesion such as an arterial anomaly must have existed for a long time, the finding of these vascular tumors must have been entirely accidental. Clinically, they do not differ in any way in the symptoms they produce from those commonly caused by lesions in the region involved.

The important facts are how to recognize these lesions and what to do for them surgically. With regard to their recognition those interested may refer to the original articles which are well illustrated. The evidences considered essential for the diagnosis of an arteriovenous aneurysm have been: marked fulness and enlargement of the veins of exit; an increased size and tortuosity of the artery entering the snarled mass of vessels. Without the enlarged veins on the cortex or elsewhere, one cannot assume the existence of an arteriovenous fistula.

The venous trunks draining the aneurysms (when located in the cerebral hemisphere) nearly always connect with the external venous circulation through the rolandic and sylvian veins, and when these form a continuous channel, as they usually but not always do, both show the maximum effect of the arteriovenous connection. This is evidenced by their dilatation throughout their course. The degree of involvement of the contiguous veins varies in individual cases. At times other nearby veins may be almost unaffected.

Because the external venous system carries the outflow of blood from most of the arteriovenous connections, the characteristic venous changes are on the surface of the brain. This makes it possible to detect these cases easily at operation. Since the veins are free from all accessory coverings, it is possible to see through their walls and detect the pulsing red, arterial blood against the background of black, venous blood.

In many of the cases of arteriovenous aneurysms located in the cerebral hemispheres, there are congenital abnormalities of the venous system on the exterior part of the hemisphere. In one case, the rolandic vein arose directly out of the brain substance, having its origin in the aneurysm. In two other cases, the rolandic vein did not exist as a well defined venous channel.

The degree of venous engorgement is dependent on the size of the arterial supply and on the size of the mass of vessels, or rather on the size of their lumens, which form the arteriovenous communication. A large angioma with many vessels takes some of the brunt of the arterial force off the venous channels of exit. A small angioma, or a short connecting link, throws all the arterial force directly into the veins. The smaller the artery and the smaller the lumen of the arteriovenous communication (i. e., the larger the number of vessels intervening between the artery and veins), the smaller will be the venous channels which drain the aneurysm. Conversely, the larger the artery and the larger the lumen of the arteriovenous connection (i. e., the smaller the mass of vessels between the artery and vein), the greater will be the effect on the veins emerging from the aneurysm. Many large veins, therefore, indicate a small angioma or a free end-to-end communication between an artery and a vein. Smaller veins indicate a larger angioma and an arteriovenous communication that is relatively well compensated, i. e., nearer the function of the capillary system. Doubtless there are many smaller arteriovenous aneurysms with a small arterial inlet and relatively small veins of exit which could not be established as abnormal. A thrill could not be felt in these, nor could arterial blood be seen pumping into them.

A mass of vessels forming the connection between the artery and veins, i. e., the so-called angioma, when such is present, is usually in the depths of the brain, but this is not necessarily true. The gross character of the mass of entwined vessels is also variable; some look exactly like arteries, some like veins and some like a combination of arteries and veins.

The arteries supplying the mass of vessels have always been larger than normal; some have been extremely tortuous. In the only case of the series in which the artery was observed, the middle cerebral artery was enlarged all the way to the internal carotid artery. It was not tortuous, nor was its enlargement excessive—perhaps from one-third to one-half larger than the corresponding vessel of the opposite side. The arterial change, while always definite, is never comparable to that of the veins, which is really the characteristic part of the picture of arteriovenous aneurysms.

With regard to the treatment of arteriovenous aneurysms, Dandy states that occasionally spontaneous recovery in cases of arteriovenous aneurysm has been reported, but there is little real hope for such a successful outcome either in the brain or elsewhere in the body. The only way to cure an arteriovenous aneurysm is to ligate the entering artery or to excise the vascular tumor. But the radical attempt at cure is attended by such supreme difficulties and is so excessively dangerous as to be contraindicated except in certain selected cases. It has never seemed fair to make this attempt except on the insistent demand of the patient who fully understood all phases of the situation. Moreover, there are ways of helping the condition with little if any risk, and this, in many cases at least, seemed more to be recommended than the attempted extirpation of the vascular mass.

As in most cerebral lesions, however, each case should be considered a law unto itself. There are large aneurysms and small ones; those which are mostly arterial, others mainly venous; some are superficial, others deep; some are in highly important areas of the brain, others in portions largely silent. All of these factors, and finally the patient's wishes in the matter must be weighed. An aneurysm in the left cerebral hemisphere in a right-handed person is surely noli-me-tangere under all conditions. Any attempted cure, even if successful, would almost inevitably result in disturbances of speech or motor power, or of both. Aneurysms of the arteries of the corpus callosum do not offer any greater surgical possibilities.

There is more reason to attempt to cure a patient who has an arteriovenous aneurysm in the right cerebral hemisphere. In most of these cases the arterial connections with the lesions are provided by branches of the middle cerebral artery. Should the aneurysm be postrolandic, the arterial connection can be ligated without injury to the motor function. In one case this could be done without great difficulty. After ligating the arteries supplying the angioma, excision of the vascular bed was easy and relatively bloodless. In another case, in which the anterior pole of the right temporal lobe was covered with great tortuous and distended veins, the anomalous vessel was located and ligated with a silk ligature. It arose from the middle meningeal artery just beyond its origin from the internal carotid. Following this the mass of distended venous trunks collapsed. This patient has remained well for three and one-half years; he has had only one convulsion (a year ago), whereas before the fistula was closed, convulsions occurred every month. There is no assurance of a permanent cure of epilepsy even after the cure of an aneurysm. The cerebral defect will always remain at the site of the old lesion, and any lesion of the cerebral hemisphere will always remain a potential source of convulsions. The convulsions, however, will be far less frequent and more easily controlled with an inactive lesion than with one which is active.

In two of the cases in which the aneurysm was located in the rolandic and prerolandic areas, palliative treatment was tried, the internal carotid artery being ligated in the neck. In one case there have been no attacks, either petit or grand mal, since the operation (two and one-half years). The other patient, roentgenograms of whom showed an extensive calcification, was free from attacks for a year, after which they gradually recurred with practically the same intensity and frequency as before the operation. As the latter patient was 47 years old, the carotid was at first only partially ligated with a band of rubber tissue. Three weeks later, it was found to be totally occluded by a thrombus.

A subtemporal decompression and excision of an extracranial aneurysm (of the scalp) were performed in another patient who was losing vision because of a long standing papilledema. His headache was relieved, but his condition otherwise was unchanged.

An arteriovenous aneurysm in the cerebellum had caused hydrocephalus. This was relieved by a bilateral cerebellar decompression which, performed as a routine, remains after a cerebellar exploration. The decompression remained full and tight until it was later relieved by ligation of the left vertebral artery in the neck. At operation, the intracranial portion of this artery was found to be several times its natural size, and this led to the belief that it supplied the arterial connection to the aneurysm. When exposed between the atlas and occipital bone, this great tortuous artery was just as large as the intracranial portion. Following its ligation, the bulging decompression subsided and has remained in this condition. Although the neurologic symptoms have greatly improved or disappeared, mental disturbance still persists.

Ligation of the vessels, whether internal carotid or vertebral, can hardly result in a cure. Only an amelioration of the signs and symptoms can be expected, and doubtless even this cannot be permanent. The precedent for such ligations is the treatment of arteriovenous aneurysms between the carotid artery and the cavernous sinus. Following ligation of the internal carotid artery for these traumatic aneurysms, there is much improvement but usually not a cure. The collateral supply through the circle of Willis militates against a cure; it is fair to presume that the same free collateral circulation will preclude the cure of intracranial aneurysms.

Venous abnormalities in the brain are described as follows: The venous irregularities were of varied form. In five instances, the sylvian vein was the site of the abnormality. In most of the cases a number of anomalous veins were spread over the frontal, temporal and parietal lobes and were tributaries to the sylvian vein. It is not an infrequent condition in presumably normal brains for the rolandic vein to be either absent or relatively insignificant, instead of being

the outstanding vein of the hemisphere. Such conditions merely indicate the great normal variations of the venous system that occur in the brain and that still appear to be of no significance.

In none of the venous deformities is the size of the vessel abnormal; rather, it is the number, shape and distribution of the vessels that are abnormal. In this respect the venous changes differ from those of the arteriovenous aneurysms in which the size of the vessels is so striking. In addition to the great size, the venous component of arteriovenous aneurysms also shows great abnormality in shape, number and distribution. One can always be sure that the lesion cannot be arteriovenous if the veins are of normal size. Moreover, in venous anomalies, aneurysms or tumors, one never sees in the veins exposed at operation pulsating red blood, which is characteristic of arteriovenous aneurysms.

In some instances, the deformation of the cerebral convolutions is even more striking than that of the veins. Many of the convolutions are of relatively enormous size and lack the characteristic shape. They appear like masses of brain tissue, improperly formed. In such cases, doubtless both the veins and the brain tissue are wrapped up in the same fundamental maldevelopment. Another example of a vascular abnormality is dependent on the primary embryonic cerebral defect; the sylvian vein received a branch from each side of the cyst and passed along the sylvian fissure as a bifid trunk. In this case the temporal lobe did not have any attachment to the parietal lobe, except in the depth of the brain.

In one case there was a venous plexus in the sylvian fissure of each side and eight or ten additional small plexuses in the pia-arachnoid over both cerebral hemispheres.

Cerebral and venous deformities are, therefore, commonly associated. The one may cause the other, or they may be entirely independent and referable to a faulty general development in the embryo. In three cases tremendous vascular deformations were present, in that many vessels of fairly uniform size radiated from the sylvian vein. In such cases the vascular bed becomes a space-occupying lesion of considerable size and doubtless acts as a tumor, for in each instance the epileptic seizures were focal and began in the face, i. e., the part of the motor area nearest the tumor. Surrounding this vascular bed, there is always a pool of fluid in the subarachnoid space, and usually the leptomeninges over the affected areas are thickened and often opaque. The brain tissue beneath is also softened and the convolutions atrophied as compared with the surrounding unaffected brain tissue. In another case, ventriculograms demonstrated that the vascular bed had indented the body of the lateral ventricle. As seen at operation, the network of vessels was so dense that the underlying brain was entirely obscured.

Another abnormality not infrequently seen is the tortuosity of veins. This is shown in a series of anomalous veins seen on one occasion. Not infrequently, one sees many of the smaller terminal veins tortuous when the larger trunks are not affected. In a recent case of congenitally deformed brain, a series of eight or ten such venules diverged from the center of the frontal lobe, some going to trunks entering the sylvian, or rolandic veins, and others to frontal veins passing to the longitudinal sinus. Tortuosity of some of the smaller veins was also present. Here there was also a diverticulum (venous aneurysm of the superior longitudinal sinus).

In looking over the histories and operative notes in these cases of venous abnormalities one finds that Dandy made no attempt to remove any of them except a venous aneurysm arising from the longitudinal sinus. Since all of these congenital venous anomalies were associated with defects in the cerebrum, the operator wisely decided that removal would accomplish nothing.

Much more can be done when a cyst with an angioma in its wall is uncovered. "Of all the tumors of the vascular system of the brain angiomatous cysts offer the best prognosis. It may almost be said that as a type and eliminating individual exceptions, they offer more than any other tumor of the brain. Usually the cyst is large and the tumor small. The tumor is embedded in and projects from some point in the wall of the cyst. Always relatively small, compared to

other tumors, it may actually be so small as to escape casual observation. There can be no doubt that in many of the so-called simple cysts a tiny angioma will be found on more careful inspection. The solid tumor is flat or rounded; it varies in size from a few millimeters to 2 or 3 cm. in diameter. Its surface is smooth and appears to be covered by the smooth lining of the cyst, but the cyst is not lined with epithelium. On sectioning the wall, a minute, insignificant looking, reddish brown lesion will frequently be found to cause the relatively enormous cyst. In the gross appearance, the intracystic angiomas are usually mistaken for gliomas. A careful gross inspection will usually make the differential diagnosis. An angioma is fibrous and acellular, and the larger tumors are usually compressible and spongelike. In a fixed specimen, the tumor has changed from reddish brown to black, because of the old fixed blood in the tumor. A glioma is softer, more cellular and consequently more friable. Cells of the latter easily scrape off on the edge of a scalpel. It is noncompressible and its color a more uniform brown in the fresh specimen and a less intense brown or black when fixed. The content of cysts, whether of angiomatous or of gliomatous origin, is apparently the same. In either type, the fluid is clear lemon or orange yellow, and a white coagulum is present at the bottom of the cyst. The reason for the cyst formation in these tumors is not known."

The reviewer wishes to state that he believes that grossly it must be difficult to differentiate the tiny angioma from a nubbin of gliomatous tissue such as is commonly seen in the wall of a cystic glioma. A frozen section might give valuable information. However, the proper procedure is the same in either case, namely, the extirpation of the tumor tissue as widely and as completely as possible.

From his experiences with five cases of cavernous hemangioma, Dandy believes that the gross appearance of these lesions is characteristic. It is spongelike and compressible because of the large spaces that one can usually see with the naked eye. It is always well circumscribed, but never encapsulated; its color is reddish-brown because of the contained blood. Some of the tumors are soft, others firm, depending on the size of the blood spaces and the amount of connective tissue. Although they have a good venous and arterial blood supply, neither is excessive, and neither is disproportionate to the other. When the tumors are opened at operation, they bleed freely and in proportion to the size of the cavernous spaces and the entering arterial supply. The bleeding is principally venous, though on the confines of the tumor the arterial bleeding may give some trouble. The tumors do not pulsate. The size of these tumors varies from minute areas to a mass larger than one's fist. Just as is true of angiomatous cysts, or for that matter any kind of tumor, the only satisfactory treatment is extirpation, when this is possible. Some tumors lend themselves readily to removal; others present difficulties which are insuperable.

While Dandy realized the relationship of angiomas of the brain to angiomas and other congenital lesions elsewhere in the body, since most of his material was obtained at operation he can contribute nothing to a better knowledge of the relationship between angiomas of the brain and cysts of the abdominal organs. It is certain that none of his intracranial angiomas were associated with similar lesions in the retinas.

GRANT, Philadelphia.

THE DISTRIBUTION AND PHYSIOPATHOLOGY OF SWEAT SECRETION. L. GUTTMANN and C. F. LIST, Ztschr. f. d. ges. Neurol. u. Psychiat. **116**:504 (Oct.) 1928.

Guttmann and List investigated sweat secretion by the method recently suggested by Minor. This method is as follows: The body or the part of the body to be investigated is painted with a solution composed of pure iodine 15.0; oleum ricini 100.0; spiritus vini 900.0. After the solution dries, the skin is sprinkled with fine rice powder and starch is pressed into the skin. The excess starch is blown away with a fan. Various sweat-producing substances are then administered. On the appearance of sweat in the areas under observation there occurs an iodine-

starch reaction which appears as a black-brown coloration, the nonsweat-secreting portions remaining colorless. Guttmann and List, after preparing the parts to be observed, gave thin patients 15 grains (0.97 Gm.) of acetylsalicylic acid and hot lime-blossom tea. The secretion of sweat was then observed. In a few cases, the experiments were repeated and pilocarpine was administered.

Minor has observed the secretion of sweat under normal and pathologic conditions by his method. He observed sweat disturbances in more than 50 per cent of all organic nervous cases. In normal persons, Minor found sweat secretion was cut off at the nasolabial fold, but Guttmann and List observed it over the nose itself. The normal sweat areas over the head are the temporal and the occipital areas. These areas coalesce with one another on intense sweating, and also with the frontal area. The parietal area, however, remains relatively free from sweat in normal persons.

Complete paralysis of the motor and sensory nerves is accompanied by anhidrosis which goes parallel with the sensory disturbance. The anhidrosis covers an area which corresponds just about with the analgesia, while in the area of thermanesthesia hypohidrosis is found. Foerster has shown that stimulation of a peripheral nerve by faradic current produces a hyperhidrosis which is spread out not only in the area of the distribution of the nerve, but also in the territory of neighboring nerves. This is seen also in nerve paralysis, where, around the area of the lesion, there may be an area of hyperhidrosis. This is brought out by the pilocarpine reaction, and is due to an increased irritability of the sweat fibers in the zones surrounding the areas of paralysis. This is seen also in areas of hyperesthesia around areas of anesthesia in sensory paralyses. In these areas of hyperesthesia there is also increased sweat secretion, and this "perilesional hyperhidrosis" is analogous to the other vegetative hyperfunctions in paralyzed nerves, such as increased growth of hair.

In partial lesions of the peripheral nerves several types of sweat secretion are noted. In the first group are cases of motor paresis with sensory disturbances: (*a*) with hyperesthesia; (*b*) with hyperpathia. Guttmann and List quote two cases, one with a partial paralysis of the median nerve with a hypohidrosis in the distribution of the median nerve, and a second case of paralysis of the radial nerve in which the cut ends of the radial nerve were sewn together with a resultant hypesthesia for touch, spatial sense, fine temperature differences and finest pain stimuli, while for somewhat stronger pain stimuli there was a hyperpathia. In this case, there was a definite hyperhidrosis in the region of the left ramus superficialis which readjusted itself with recovery. Such an observation had been recorded by inspection by Foerster who found that the return of the sweat fibers after sewing a cut nerve was parallel with the restoration of the pain fibers. On the other hand, it is not improbable that the complete restoration of the normal sweat fibers is dependent on the complete regeneration of the other sensory modalities which appear to have a regulating influence on the sweat secretion. In the second group of partial peripheral nerve lesions, namely, cases of motor paresis without sensory disturbances, there are never any sweat disturbances except as regards the face. In pure irritation processes without motor paresis, as in neuralgias, hyperhidrosis is usually found. This may be produced in two ways: (1) by direct stimulation of efferent sweat tracts, in which case the hyperhidrosis is confined to the distribution of a particular nerve, or (2) by irritation of an afferent tract in which case the hyperhidrosis is more widespread and not confined to the distribution of a single peripheral nerve.

The sweat fibers to the face behave differently from those to the extremities. While in the latter a total peripheral nerve lesion is accompanied by anhidrosis, this is not the case with the facial nerves, sweat fibers to which are carried by the cervical sympathetic nerve. Guttmann and List found complete unilateral anhidrosis in a sympathetic paralysis. There is some question as to the relation of the facial and trigeminal nerves to sweat secretion. Some, as Dejerine and Minor, assume there are sweat fibers in the facial nerve, while others believe the trigeminal is the chief sweat conducting nerve. Guttmann and List believe that the facial nerve is the chief carrier of sweat fibers, for in six cases of facial palsy

they were always able to demonstrate a hyperhidrosis in the area of nerve paralysis. As to the trigeminal nerve, in a case of complete unilateral sensory and motor paralysis of the trigeminal nerve, due to carcinomatous metastasis in the gasserian ganglion, no disturbance of sweat secretion was demonstrable. Intracranial excision of the second and third divisions gave smiliar results. An alcoholic injection of the second division, however, in a case of trigeminal neuralgia, with a resulting anesthesia in the area injected, resulted also in hypohidrosis. Guttmann and List therefore conclude that there are no sweat fibers in the trigeminal roots, gasserian ganglion and intracranial divisions of the nerves, but that sweat fibers are present in the peripheral branches, which mix with the trigeminal branches from the carotid plexus, and that the sweat fibers in the facial nerve reach the skin by means of anastomoses of the facial with the trigeminal nerve.

The question remains as to the relation of the anterior and posterior roots to sweat secretion. Foerster found a marked sweat reaction extending over several dermatomes on stimulation of an anterior root. Thus, on stimulation of the tenth dorsal anterior root, he obtained responses in dermatomes from the seventh dorsal to the fifth lumbar. On stimulation of the tenth dorsal posterior root, however, the sweat secretion ceased in the tenth dorsal dermatome, and was present in all the others. Foerster therefore concluded that "an anterior root carries sweat-exciting fibers for several dermatomes, while the posterior root contains sweat-inhibitory fibers only for the one dermatome it supplies." He demonstrated this further by stimulating the fifth dorsal anterior root and obtaining sweat secretion in the seventh to the tenth dorsal dermatomes, and later in the fourth to the tenth dorsal dermatomes.

As for the spinal cord, the spinal sweat centers show a marked regional and segmental arrangement. A complete transection of the spinal cord produces anhidrosis below the level of the lesion. This means that the cerebrofugal sweat tracts possess an inhibitory as well as an innervating function. The cerebrofugal tracts in the spinal cord lie in the lateral columns close to the pyramidal area, so that a unilateral anterolateral cordotomy produces no effect on the sweat secretion, provided the neighboring territory of the lateral horns remains intact. Guttmann and List could find no cases of bulbar and pontile sweat disturbances, but there were numerous cases of subcortical lesions in the basal ganglia with sweat disturbances. In cases of parkinsonism they found generalized hyperhidrosis and also hypohidrosis. In capsular lesions, if seen early, they could always demonstrate hyperhidrosis of the contralateral side. As to cortical lesions, there is no uniformity. Bechterew obtained sweat secretion of the contralateral side in cats on stimulation of the cortex. Winkler found only slight sweat secretion on stimulation of the motor area in cats, but greater secretion on stimulation of the medial part of the base of the frontal lobe. Karplus and Kreidl obtained no effect on stimulating the cortex after a section through the hypothalamus, and others have failed entirely to produce sweat secretion on stimulation of the cortex. In human pathology, Goldstein, on the basis of cortical injuries, designated the motor area as the sweat center. The experience of Guttmann and List with cases of cortical injuries in Foerster's clinic forces them to give a rather extensive localization of the sweat center in the cortex. They found disturbances of sweat secretion in lesions of the parietal lobe, in the precentral and postcentral convolutions, in the first temporal convolution, field 6*a* and 6, and in the previsual area. No sweat disturbances were found in lesions of the most anterior part of the frontal lobe and of the occipital lobe.

Sweat innervation is mediated by at least five neurons: (1) from the cortex through the internal capsule to the subcortical ganglia, apparently the hypothalamus; (2) from the hypothalamus to the vegetative oblongata nucleus; (3) from this latter nucleus, some crossed and some uncrossed fibers to the lateral horns of the spinal cord—the decussation takes place apparently at the level of the pyramidal decussation; (4) from the lateral horn through the anterior root via the ramus communicans albus to the peripheral ganglion; (5) from the sympathetic ganglion through the gray ramus communicans to the peripheral nerves of the sweat glands.

The entire system of sweat innervation is, therefore, made up of a platform of reflex arcs: (1) one from the cortex, which is excited particularly by psychic effects; (2) a subthalamic arc, subject to thermic impulses; (3) a bulbar arc, subject to taste stimuli; (4) a spinal arc, which is activated by sensory cutaneous stimuli.

ALPERS, Philadelphia.

CEREBRAL HEMORRHAGES IN YOUNGER PEOPLE AND THEIR DIFFERENTIAL DIAGNOSIS. OTTO MARBURG, Deutsche Ztschr. f. Nervenh. **105**:22 (Sept.) 1928.

Cerebral hemorrhage is usually looked on as an expression of severe arterial changes in advanced age. The occasional cases of cerebral hemorrhage in younger people have not so far been sufficiently studied. Marburg recently had occasion to study several cases in younger people with a nearly apoplectiform onset, which made the diagnosis of hemorrhage most likely. This diagnosis was not always easily made at the beginning. Encephalitis or tumor had to be considered. Histologic studies of one case permitted the author to recognize the full extent of this disease.

The cases studied were in patients who ranged between the ages of 30 and 50. In one case only, the youngest patient of the series, a woman aged 33, was the diagnosis of hemorrhage most probable. She had had considerable hypertension for a period of ten years for which no cause could be found. In the other cases, previous to the onset, nothing pathologic was found in either the heart, the blood vessels or the blood pressure.

CASE 1.—A man, aged 43, held a position of great responsibility and worked for more than fourteen hours a day; he was never sick; there was no sign of syphilis, and he had two healthy children. A few days before the onset, he complained of a dulness in the head, but continued to work. One afternoon, the headache suddenly increased and an hour later it was followed by an epileptic attack, resulting in a left-sided paralysis of the arm, leg and face. Marburg found the patient unconscious; the pupils reacted; there was hyperemia of the fundi; there was a definite pyramidal disturbance on the left, and to a lesser extent on the right side; the pulse rate was 50; breathing was stertorous; there was no increase in temperature; the white cell count was 10,000, mainly neutrophils. Various medications to reduce the intracranial pressure brought no definite change in the patient's condition. A right-sided decompression was done over the motor area. The brain was under considerable pressure, the gyri were flattened and the sulci were obliterated. The patient died a few hours after the operation.

A postmortem examination disclosed a large right parietal hemorrhage just under the cortex; a similar hemorrhage was found in the same area on the left side and near to it were many very small hemorrhages. Histologically, near the large hemorrhage, the pia was soft, but in a few places there was considerable hemorrhage. The pial blood vessels were unusually dilated and engorged with blood, but there was no alteration in their walls except that they were thinned out and the intima cells were swollen. The pia was not adherent to the cortex. The cortex was edematous, especially perivascularly. In some places it gave the impression of a perivascular disintegration. The capillaries showed swelling of the intima cells. The smaller arteries also failed to show an alteration of the walls. There was a beginning of glial irritation. In the white matter there was considerable edema, with a few small hemorrhages. Here the glia was markedly swollen and formed a thick lacunar net. Some places gave the impression of sclerotic structure or tumor, without any definite indication of a tumor. There was a dense fibrous glia reticulum with scattered nuclei. The blood vessels here, too, showed only a swelling of the intima cells. In a few places a perivascular effusion was noted. The thick lacunar glial net passed over to the gray matter of the cortex. In this net were perivascular hemorrhages. A number of arterioles were surrounded by a circle of red blood cells. Many such blood vessels, lying close together, gave the impression of a purpura cerebri. In such hemorrhages

were noted already necrotic changes of the glia net, paling, rarefication and necrosis of the vascular walls. Here some blood vessels were found entirely occluded. The large hemorrhage destroyed all tissue. The area adjacent to the hemorrhage gave the impression of a marked malacia, without any glial reaction. In a few places blood could be seen definitely coming from the blood vessels, the walls of which appeared torn. The glia stain gave the impression of a dense glial net, which in some places stopped sharply at the hemorrhagic area, and was studded with minute hemorrhages. This was more noticeable in the white than in the gray matter. It was noted that the vascular walls in the hemorrhagic areas did not stain normally; the media and adventitia failed to show a differentiation; they appeared more homogeneous, although they could not be spoken of as sclerosed. The ganglion cells appeared normal, while the nerve fibers, especially in the hemorrhagic areas and hence in the white matter, appeared markedly affected. The axis cylinders were swollen and in some places torn, especially near the hemorrhagic area.

Commenting on the histologic observations, Marburg rightly observes that from the pathologic anatomic observations a causative diagnosis cannot be made since no postmortem study was made of other organs. Conclusions can be drawn only from clinical examinations. It is to be noted, however, that there was no indication of a cardiorenal disturbance, and marked hypertension can be excluded. The point noted about the cerebral blood vessels was thinning of the walls; evidently they could not withstand increased activity. In the hemorrhagic areas the blood vessels showed marked changes; the walls were homogeneous, and in some places distinctly necrotic; whether this process was primary or secondary to the the hemorrhage or edema the author is unable to say.

The hemorrhage could be considered as the result of a vascular weakness ab ovo. The hemorrhage was strictly vascular and not a diapedesis, and the most important point to be noted is that it was localized mainly in the white substance.

In a former work, through Shibuja (*Arb. a. d. neurol. Inst. a. d. Wien. Univ.* **26**:253, 1924), Marburg has shown that hemorrhage in the white substance in consequence of the swelling may be the cause of prolapse of the brain. The hemorrhage alone is sufficient to cause an intense edema and lymph stasis. Marburg suspects this to have been the cause. With a comparatively mild beginning, the increase in the intracranial pressure soon brought on secondary hemorrhages, and the purpura cerebri took place in this way. It is always the hemorrhage in the parenchyma that is responsible for the swelling manifestations, the lymph stasis and the general edema. With an opening in the skull, a prolapse will develop, otherwise it will result in marked rise of intracranial pressure.

Case 2.—A man, aged 50, with a negative previous history except that for some time he was subject to considerable excitement, for a few days had headaches and then suddenly fell unconscious, and developed a complete left paralysis. There was no cardiorenal involvement. A tumor or an inflammatory process was considered as the probable diagnosis. When Marburg saw the patient he found, in addition to the left-sided paralysis, a homolateral ptosis, a mild contralateral facial paresis, and a bilateral Babinski sign. A multiple process was evident. There was a slight edema of the papilla. Alternations between mild somnolence and marked unconsciousness recurred for a long period; after each spell of unconsciousness the somatic manifestations became more aggravated, without new lesions being detectable. The temperature was 90.7 F. The patient died. A postmortem examination was not obtained.

Case 3.—A woman, aged 33, suffered from uterine hemorrhages for ten years, for which no cause was found other than an excessive hypertension. There was present a familial disposition for this. Without any premonitory signs she had a sudden stroke, with complete unconsciousness and signs of intracranial pressure, bradycardia, vomiting and hyperemia of the papilla. After some medication, when some improvement had set in, a diagnosis was made of a large left-sided hemorrhage involving the motor speech area. A careful examination disclosed that the other side of the brain was also involved. The patient died two and one-half days after the attack. A postmortem examination was not made.

Case 4.—A man, aged 40, suddenly developed cerebellar disturbances (his father, at 39, suffered from similar attacks), with left facial paresis, increased deep reflexes, bilateral Babinski sign and diminished abdominal reflexes. The systolic blood pressure was 165; a white cell count showed 4,000 cells. This ruled out encephalitis, and Marburg diagnosed the condition as multiple small hemorrhages. Therapy calculated to reduce the blood pressure brought complete recovery from the symptoms and also from the subjective complaints, such as headaches and dizziness. (Multiple sclerosis should have been considered as a possible diagnosis in this case, Abstracter.)

In discussing the differential diagnosis in these cases, Marburg calls attention to the importance of excluding a possible tumor. Headache of a few days' duration, symptoms of general intracranial pressure, minimal papillary hyperemia, and focal signs when present should lead one to think of a hemorrhage in a latent glioma. A multiplicity of lesions, especially with an apoplectiform onset, must exclude tumor. The differential diagnosis from encephalitis offers greater difficulties. Premonitory headaches occur in both. Encephalitis does not usually have an apoplectiform onset, but occasionally it does. Both may show a leukocytosis. In encephalitis, especially in the lethargic type, the blood shows a lymphocytosis and a greater cell increase; there is an increase in temperature; the onset is not so sudden and is usually not marked by intracranial pressure, and the somnolence is more characteristic.

Marburg is of the opinion that the pathology of the blood vessels was the result of some unknown infection or of a nephrosis. Three of the patients gave a history of grip three weeks before the onset of the disease, which passed off without leaving any visible after-effects. Pollak and Rezek, from Marburg's Institute, report similar observations in such cases (*Virchows Arch. f. path. Anat.* **256**:683, 1927). In the investigated case Marburg is inclined to assign the thinning of the vessel walls to a congenital disposition; the inciting moment of the hemorrhage he is unable to state; overwork, excitement or some other unknown factor causing a sudden rise in the pressure may bring about the first hemorrhage. When this takes place in the white matter it is followed by marked edema; this again predisposes to further hemorrhage, and often follows a symmetrical course.

Bernis, Rochester, N. Y.

The Pathogenetic Relations Between Tumors of the Spinal Cord and Syringomyelia. Eugen Kirch, Ztschr. f. d. ges. Neurol. u. Psychiat. **117**:231 (Nov.) 1928.

According to Saxer and Schlesinger, syringomyelia does not represent a nosologic entity; different causes are concerned in the occurrence of these cavity formations in the spinal cord. Genuine syringomyelia is a more or less longitudinally extending cavity separated from the central canal. In some of these cases, the occurrence of the cavity is the result of a degenerative or inflammatory destruction of the substance of the spinal cord. In others, a traumatic etiology is possible. Another group of cases of syringomyelia shows the cavity formation in the spinal cord in combination with tumors or tumor-like formations. This includes a large group of cases, especially if those cases with a small gliosis and a central cavity are included. It is with this group that Kirch is particularly concerned. He observed cases of intramedullary tumors of the spinal cord with cavity formation, and drew a parallel between the formation of cavities in these cases and of cysts in tumors of the brain. Cysts may develop in tumors of the brain in one of two ways: they may develop within the tumor itself as the result of degeneration and destruction of the tumor tissue resulting in a cyst within the tumor, or they may occur wholly outside the tumor as Lindau has shown. In these latter cases, the cyst is the outstanding feature, and the tumor is small. In Lindau's conception, the cyst formation in these cases is due to circulatory disturbances caused by the tumor with edema of the brain and a resulting destruction of the brain tissue, wholly apart from the tumor. These fundamentally different cyst or cavity formations hold also for syringomyelia in tumors of the spinal cord.

Here, too, the cavity formation is both in and outside the tumors as in the case of cysts of the brain.

Kirch does not include in the first group, that of cavity formation within the tumor, the cases of primary gliosis with a central cavity. Only true tumors are included, and the first case which he describes is one of syringomyelia in a man, aged 43, which at necropsy showed a central cavity formation within a tumor stretching the entire length of the cord. No remnant of the central canal was left. The tumor itself was rich in cells and was classified as a gliosarcoma. That the case was one of tumor and not a gliosis is demonstrated: (1) by the richness of the cells as contrasted with cell poverty in a gliosis; (2) by the fairly well defined boundaries of the growth as contrasted with the gradual distinction from the rest of the cord substance in cases of gliosis, and (3) by the fact that in this case the tumor had extended to the meninges of the spinal cord and involved them for some distance, a thing which is impossible in a mere gliosis. Kirch looks on the tumor formation in this case as primary and the cavity formation as secondary, originating through necrosis and destruction in the inner part of the tumor. Only scattered cases of a similar nature appear in the literature. Zander reported a "glioblastic sarcomatosis" which had grown in the center of the entire spinal cord and spread to the meninges in a boy, aged 8. There was no cavity formation, but at certain levels of the tumor there were unconnected cavities, especially in the middle cervical, upper thoracic and lower lumbar cord. Besides these, there were several smaller cavities. Zander mentioned syringomyelia in this case. Schultze (1878) reported the case of a boy, aged 18, with a pencil-like tumor extending the length of the cord, with central cavity formation and involvement of the meninges. The tumor was looked on as a telangiectatic and myxomatous gliosarcoma, and Schultze considered the cavity a malformation. Raimond and also Thiele described similar cases of pencil-like tumors extending the length of the cord, with central cavity formation. Raimond's case was a glioma, and Thiele's was a "neuroepithelioma gliomatosum microcysticum." The necrosis was limited to the dorsal and lumbar sections of the cord. Other cases of glioma with central cavity formation have been reported, but the tumors in these cases have not extended the entire length of the cord. Many cases of tumor which extend the entire length of the cord without cavity formation have been reported. In all the cases cited, the tumor was a glioma of one sort or another. Nonne, on the other hand, described a tumor of the spinal cord, a round and spindle cell sarcoma, which spread the entire length of the cord and into the medulla where it showed a cavity formation extending to the third cervical segment. Apart from the case of neuro-epithelioma of Thiele, none of the tumors were of epithelial origin. Kirch's second case, however, consists of a metastatic carcinoma extending from the fifth to the twelfth thoracic segment with a central cavity formation which was not connected with the central canal. The primary carcinoma had been removed from the ovary ten months previously. The second case is of interest for two reasons: (1) the occurrence of carcinoma within the substance of the spinal cord itself, and (2) the occurrence of syringomyelia in connection with it. The view that the spinal cord is never involved by carcinoma was disproved by Miller, who described a case of primary carcinoma of the lung with metastasis to the spinal cord. Taniguchi, Sadelkow and Nonne have reported other authentic cases involving the segments from the tenth dorsal to the third lumbar, the fourth lumbar and the upper cervical cord, respectively. With regard to the cavity in Kirch's case, it is important to note that it was not connected with the central canal and did not lie in the cord substance, but lay in the cancer metastasis itself and was surrounded by tumor cells.

The second group of cases of syringomyelia includes those of tumor formation with cavities within the substance of the spinal cord itself. These are parallel to cyst formation in the brain as described by Lindau and Schley. The tumors of the cord with this second type of cavity formation differ from those of the first group in that the size and extent of the tumor are of no importance. Small tumors are often accompanied by extratumor intramedullary cavity formation.

Many different histologic types of tumors have been described with this formation: gliosarcoma, angiofibroglioma, neuro-epithelioma gliomatosum, hemangioma (capillary and cavernous), spindle cell sarcoma and carcinoma. An examination of these tumors reveals that most of them are extremely rich in blood vessels, and that therefore tumors rich in blood vessels are most prone to develop this type of syringomyelia. This fact is in unison with the brain cysts in which hemangiomas are important. Another fact of some importance in the tumors of the spinal cord mentioned lies in the almost constant presence of numerous vascular changes. Almost every investigator has reported more or less pronounced hyaline changes in the vessel walls with narrowing of the vessel lumen, and often with complete obliteration of the lumen. Endothelial changes with complete obliteration are often noted. In places, thromboses occur. These vascular changes cause a blood stasis which is important in the development of the syringomyelic cavity. Areas of edema occur within the tumor, around the thrombosed vessels. These occur also outside the tumor, in the spinal cord substance itself. Schüle found localized edema around his tumor, and small areas of softening. Friedrich and Stiebler saw this in the medulla oblongata around a hemangio-endothelioma. Around the tumor, the medulla was swollen and edematous, with small cyst formations surrounded by glia cells. They believed that such cysts were the result of edema, stasis and degeneration of the nerve tissue. Kirch states that the glial formation seen around syringomyelic cavities is not a primary, but a secondary matter. It is not a true tumor formation, but a reactive gliosis, a glia hyperplasia caused in a purely mechanical manner by the pressure of the fluid on the surrounding nerve tissue. The glial increase is accompanied by fiber proliferation which, after a time, increases faster than the glia cells, giving a cell-poor fiber-rich capsule around the syringomyelia. This conception of the perisyringomyelic gliosis makes firmer the parallel between syringomyelic cavities and cysts of the brain. Around the latter, according to the work of Schley, there is a mantle-like glial layer of the same origin and characteristics. It consists of a capsule poor in cells and rich in fibers which are arranged in parallel rows. Kirch reports three cases of intramedullary tumors of the spinal cord with small syringomyelic cavities in the cord itself outside the tumor, surrounded by glial walls as described, and with thrombus formation in the tumor.

ALPERS, Philadelphia.

THE PATHOGENESIS OF INVOLUNTARY MOVEMENTS WITH SPECIAL REFERENCE TO CHOREA. S. A. KINNIER WILSON, Deutsche Ztschr. f. Nervenh. **106**:109 (Dec.) 1928. (From the Proceedings of the Eighteenth Annual Congress of the German Neurological Society held in Hamburg, Sept. 13-15, 1928.)

As Hughlings Jackson's pupil, I cannot be satisfied with the manner with which many neurologists associate clinical symptoms with pathologic lesions. The fundamental mistake of the clinical-pathologic method is that there are two types of clinical symptoms, such as destruction of function and dysinhibition of function; but there is only one kind of pathologic lesion which can be proved with certainty, namely, destruction. Many neurologists believe that choreic movements can be explained by a deficiency in striatum elements. Against this I may say that histologic lesions affecting paralysis can be explained only on the basis of a destruction or loss of nerve function. But the problem of all involuntary movements — as tic, epilepsy, tremor, chorea, athetosis, myoclonus, etc. — is one of physiology. It is clear that these problems can never be settled if one assigns as the cause of these movements a destroyed cell fiber system. All involuntary movements depend on a living mechanism, and the question must therefore not be: Which mechanism is paralyzed? But rather, Which mechanisms are in activity? For the problem of chorea, a careful analysis of the choreic movements is of great importance. One finds that the spontaneous movements in chorea strongly resemble those which are designated as voluntary. They appear as complicated, coordinated and serving a definite end as those of the free selective type. On the other hand, the voluntary movements of the choreic patient are often as short, abrupt and explosive as the

involuntary movements. Further, in chorea one finds definite more or less "minor signs" which point to a corticospinal disturbance. From those clinical facts the following conclusions may be drawn: The choreic disturbance manifests itself through the corticospinal (pyramidal) system; this must be in a relatively entire condition, making it possible for the hyperkinetic symptoms to appear. Yet it may show a defect in its function. Therefore, as I observed, the voluntary movements of the choreic patients do have something of the character of involuntary movements; the latter take the same route and usurp at the cost of the former. Wilson showed some pictures in a case of senile chorea which was under his observation for eleven years. The patient, a woman, died at the age of 88. The marked choreic movements affected only the right side. The postmortem observations showed that the convolutions were generally well maintained; the left postcentral convolution alone was shrunken to nearly one-half the size. The entire left corpus striatum was well preserved in all parts, even considering the age of the patient. There was no macroscopic difference between the right and left striatum. The left putamen showed microscopically as good as normal structure; the large as well as the small cells were intact. According to Wilson this case explodes the striatum theory of chorea. He would consider the atrophy of the left postcentral convolution of the greatest importance for the pathogenesis of chorea, and offered the following hypothesis: for the occurrence of choreic movements it is necessary to have a relatively integral efferent tract. Many compelling reasons suggest the assumption that this tract is the corticospinal. He has never seen a case of chorea or of athetosis in which spontaneous movements took place in association with a complete corticospinal paralysis. Such movements were never observed in man with decerebrate rigidity, and Magnus stated that it was absent in lower animals which he has decerebrated. The explanation which the author would give in the cases of chorea or athetosis is that in decerebration the motor cortex is out of function. A number of clinical facts would indicate that in cases of chorea-athetosis the afferent tracts from the cerebellum via the regio subthalamica to the thalamus and the brain cortex are involved; hence, the great importance of the postcentral atrophy in his case of chorea. Certain changes in the sensorimotor cortex have taken place which point to a disturbance in the regulation of motor stimulation of the transcortical as well as of the cerebellocerebral side. The author points out as the most characteristic trait in chronic choreic movements the inability on the part of the patient to influence voluntarily the movements or to bring them to a standstill: expressed physiologically, it can only indicate a transcortical damage of the function, and in his case he connects the loss of the transcortical control with the atrophy of the sensory part of the cortex. In this place, most likely, the activity of the cerebellocerebral component occurs. The author summarizes his conceptions as follows: Chorea and choreic athetosis represent a complex type of involuntary movements for the activity of which a motor mechanism having its seat in the cortex is required. It is not possible to assume that a single circumscribed anatomic area is responsible for developing these symptoms. Chorea is an expression of the disturbance of a system. A defect in the afferent cerebellocerebral regulation contributes to its development; the transcortical (voluntary) is very incomplete in its expression. No theory which assigns a single destructive lesion in the corpus striatum as the cause of chorea will agree with the facts. No hypothesis as to the cause of involuntary movements can be discussed which does not follow the interpretation of semiologic and pathologic principles, thanks for which are due largely to the genius of Hughlings Jackson.

Discussion.—Dr. O. Marburg: I agree with Wilson that chorea and athetosis are complicated processes, but they do not take place only through disturbance in the transcortical regulations. All lesions in the cerebellocerebral tract are capable of producing a similar disturbance without participation of the cortex. I recall a case reported by Halban-Infeld, in which a small circumscribed lesion was responsible for the athetosis, and a case reported by Herz in which a small lesion was found in the thalamus. A lesion in the end-system is also capable of producing the same; fundamentally it is concerned with a centripetal regulation disturbance.

DR. MINGAZZINI: I agree entirely with Wilson when he assumes that choreic movements depend only on disturbance of the activity of the cerebellocerebral (cerebropetal) fibers. For many years I have supported the assumption of the existence of such fiber complexes, especially on the basis of personal investigations by the absence of lacunae in sections of the brain in man, in one of which was found an aplasia of the cerebellum, as well as in those of the dog and monkey in which the cerebellum or the cerebrum was removed and in the human brain when the cerebellum was found nearly absent on one side. Now in these cases the homolateral and the contralateral transverse fibers of the pons, the fibers of the medial and lateral fifth of the pes peduncili, the anterior segment of the internal capsule and some contralateral frontal convoluntions were in places either partly degenerated or aplastic. The course of the disease process spoke distinctly for the presence of cerebellofugal (not cerebellopetal) fibers which end in the frontal lobes and perhaps connect with the pyramidal cells in the rolandic area. These cases agree completely with the pathogenesis of the movements in chorea and athetosis as given by Wilson.

DR. A. WIMMER: I agree entirely with Kinnier Wilson in his demand for the entirety of the corticopyramidal system for the appearance of choreic disturbances. The comparison of choreic and normal movements is not entirely convincing; not only is it a question that the choreic patient cannot voluntarily suppress his movements, but it concerns rather a disturbance of a succession of movements, a disturbance of transmission. I agree with Kinnier Wilson that destruction cannot give positive physiologic disturbances. Things are altogether different when it concerns a possible disturbance of transmission. One must hold to the idea that such disturbances can be effected chiefly from the corpus striatum. At any rate one must at present consider the corpus striatum as the most important "editorial place" in the brain in the bringing on of involuntary movements.

BERNIS, Rochester, N. Y.

DISTURBANCE OF THE SPINAL FLUID SYSTEM IN HEAD TRAUMA. PETER BIELSCHOWSKY, Ztschr. f. d. ges. Neurol. u. Psychiat. **117**:55 (Nov.) 1928.

Bielschowsky continues the work reported in 1924 by Foerster and Schwab. The determination of changes in the fluid system is important, because often the clinical symptoms show no focal symptoms, while there is present a characteristic general symptom-complex. This "post-traumatic general cerebral syndrome" consists of headaches, vertigo, vomiting, tinnitus, scotomas, marked lability of the circulatory system, general bodily incapacity and psychic disturbances consisting in emotional lability, tendency to depression, irritability, decrease in mental capacity and decreased concentration. These symptoms occur in a great number of cases of injury to the head, and they occur with and without fracture of the skull, and with and without evidence of focal symptoms. It is of extreme importance to determine whether these posttraumatic symptoms are due to organic injury of the brain and its membranes, or whether they are psychic in origin. Since there is a disturbance in the spinal fluid system in these cases, Foerster looks on the term "post-traumatic general cerebral syndrome" as pertinent and to the point.

Bielschowsky reports on 106 cases of head trauma with disorders in the dynamics of the spinal fluid. The routine examinations of the fluid included pressure studies, resorptive studies, transit of the fluid and encephalography. An increase of more than 200 mm. in the fluid pressure was present in 54 per cent of the cases. There was a retardation in resorption of the fluid in 58 per cent of the cases.

The encephalogram has three characteristics in traumatic cases: (1) lack of filling of the ventricles; (2) change in the form or position of the ventricles, and (3) change in the distribution of air over the cerebral surfaces (either an increase or a decrease). These characteristics may be present singly or in unison. The lack of filling of the ventricles was present in fourteen of the 106 cases. The failure of the ventricles to fill is looked on as an error of technic or as a dis-

tinctly pathologic condition. If sufficient air (from 100 to 150 cc.) has been introduced, the observation is pathologic. The ventricles can fill with only 50 cc. of air, but conclusions from this amount must be drawn carefully. Foerster considers the lack of filling when sufficient air has been introduced to be pathologic, because in these cases, when ventricular puncture is done, there is a marked dilatation of the ventricles. More important, however, Foerster has found, on operative intervention over the cisterna cerebellomedullaris, that the communication between the fourth ventricles and the subarachnoid space is obstructed by meningeal membranes or by the cerebellum being pressed down into the foramen magnum. Most cases of failure of the ventricle to fill show elevated pressure of the spinal fluid and more or less pronounced disturbance in resorption of the fluid. As a rule, the obstruction is in the foramina of Magendie and Luschka, but in cases in which the fourth ventricle is filled the obstruction is in the aqueduct. When both the third ventricle and the fourth ventricle fill, the obstruction is at the foramen of Monro.

Changes in the shape of the ventricles involve usually both lateral ventricles, but frequently implicate the third and fourth also. Besides dilatation of one or both lateral ventricles, there is often a pulling of the ventricle to one side. In these cases, the dilatation of the ventricle is on the side of the trauma. This occurs with great regularity when there is a bony defect, but also when there is a slight bony depression or a hardly visible fracture line. Often in cases with no bony change, but with a contusion of one hemisphere, there is dilatation of the lateral ventricle on the side of the contusion. Finally, dilatation of the ventricle occurs on the side of the trauma when there is evidence neither of bony fracture nor of contusion of the brain. Bilateral dilatation of the ventricles occurs almost as frequently as ventricular asymmetry. This may be slight or pronounced, giving the appearance of an internal hydrocephalus. Fairly often, there is bilateral ventricular dilatation in the posterior part of the head. Many authors look on the ventricular changes as not due to trauma, but as being caused by anatomic variations present before the trauma. In more than 1,000 cases in which encephalograms were obtained, however, Bielschowsky has been able to determine the normal contours.

The discovery of an increased amount of air over the cerebral surface is evaluated with care by Bielschowsky. A certain amount of increase over the frontal and occipital poles is normal. Of particular significance, however, are the circumscribed collections of air. These are arachnoidal cysts which have been verified by operation. They are related to a traumatic porencephaly. The basal cisterns often show an abnormal degree of filling. Frequently, in a lack of filling of the ventricles, there is an increase of air over the cerebral surfaces.

Deviations from the normal encephalogram were found in four fifths of Bielschowsky's 106 cases. The question arises then on what the changes in the fluid system rest. The chief cause is the posttraumatic meningopathies: arachnitis serofibrosa cystica adhesiva or meningitis serosa. These meningopathies cause a diffuse increase in fluid as well as cyst formations within the arachnoid, and circumscribed collections of fluid. The ventricles are occluded in these cases by adhesions in the region of the foramina of Luschka and Magendie. Fluid secretion is increased in the meningopathies and fluid pressure is increased. An increase in cells and albumin is lacking in most cases. In addition to the meningopathies, the vasopathies and the encephalopathies are of importance in causing the picture seen in the encephalogram.

Clinically, the cases fall into three groups: (1) injuries to the head without focal symptoms but with the general symptoms described; (2) cases with general and focal symptoms, and (3) cases of traumatic epilepsy.

The first group comprises 48 cases, with an increase in pressure in 24, and a disturbance in resorption in 29. The number of cases in which a normal encephalogram was obtained was 37, or 77 per cent. The second group comprised 38 cases of injury to the head with, besides the general symptoms, focal signs pointing to the cerebral nerves, the midbrain or the cortex. These were cases

of severe trauma. In 17 of them there was an increase in pressure; in 23 a prolongation of fluid resorption, and in 33 a change in the encephalographic picture. Only 5 cases showed a normal encephalogram, but all these showed increased fluid pressure and a prolongation of fluid resorption. Traumatic epilepsy occurred in 30 of the 106 cases. Epilepsy was a late result of trauma. In the cases of epilepsy, the general cerebral symptoms were less pronounced than in the other two groups. The majority of the cases of traumatic epilepsy showed severe disturbances in the ventricular system. Increase in pressure in these cases occurred 18 times, and resorptive disturbances in 10 cases. There were 26 pathologic encephalograms in these cases, of which four showed failure of the ventricles to fill.

ALPERS, Philadelphia.

CYTO-ARCHITECTONICS AND PROGRESSIVE CEREBRATION. C. VON ECONOMO. Rev. neurol. **2**:643, 1928.

A hundred years of studies on the brain have scarcely yet scratched the surface of the problem of psychic life. Until recently, the cortex was held the seat of the highest psychic function, but this conception has now been somewhat dethroned by increased acquaintance with epidemic encephalitis, showing the importance of a subcortical mechanism in the reactions and emotions. Nevertheless, the cortex is still to be considered the substratum of superior intelligence, especially for such functions as apperception, recognition, memory and reason. The cortex, after all, is an organ for the transmission of energy and as such is only a complicated reflex system, but when one considers the enormous number of cells, about 14 billion, one realizes the stupendous character of this organ and the variety of reflexes of which it is capable. Heretofore, studies of the cerebral cortex have been most intensively followed out in the realm of cell types, although such studies as angio-architectonics, glio-architectonics, fibrillo-architectonics and myelo-architectonics are not to be forgotten. The author has recently completed an intensive study of the cyto-architectonics of the cerebral cortex.

In the cortex one finds three types of cells: pyramidal, fusiform and granules. They are laid down in the cortex in six layers, and it is by differences in the width, richness and arrangement of these various layers that portions of the cortex differ from one another and may be identified by mensuration. This leaves out, however, the allocortex, which in man makes up only one twelfth of the surface and is the rhinencephalon. Needless to say, in lower animals this allocortex is much more impressive in its dimensions. In a consideration of the isocortex only, it has been possible to divide it into 107 different areas, depending on the relationship of the cellular layers to one another and the predominance of this or that type of field. It must not be supposed that these fields are strikingly different from adjacent ones in all cases, and yet in at least two instances the transition is truly abrupt. Von Economo calls attention to the change from one type of cortex to another in the sulcus of Rolando, and also at the extremity of the visual cortex near the calcarine fissure. Here the changes are exceptionally abrupt.

Although more than 100 different fields have been distinguished, these can be resolved into five different general types for purposes of comparison and for better correlation of structure and function. The first type is the agranular cortex, great in depth, with large cells, the type one finds in the precentral convolution, exquisitely motor in function. At the other end of the scale is the thin cortex, with no large cells but an infinite number of granules, which is the characteristic example of the primary sensory cortex. Between these extremes there are three grades: one approaching type 1 in its characteristics, typifying the frontal lobe; an intermediate portion, with the granules and pyramids about equally developed, characterizing the parietal area, and one in which the cortex is distinctly thinner and the granules about equally numerous, characterizing the frontal and occipital poles of the brain.

In order better to understand the function of these layers, one may take it as indicated that the afferent fibers enter the fourth or granular layer, which constitutes the receptive area; that the third layer is concerned with the association

of such impulses and their transmission to other adjacent and distant parts of the cortex, and that the fifth and sixth layers are concerned with the efferent impulses. Cajal considers the second or external granular layer as the origin of the callosal fibers and therefore an associative system. The first layer is probably a zone for the intermingling of fibers from cells immediately adjacent to one another. Thus, it can be seen that the agranular cortex is largely motor in function, and that the granular or koniocortex is largely or wholly concerned with the reception of the afferent impulses. The five types of cortex are disposed as follows: The first, or motor, occupies the prerolandic areas, a rather large extension on the mesial surface, the anterior part of the insula and the base of the frontal lobe. Moreover, there are patches of agranular cortex to be found posterior to the corpus callosum and in the hippocampal region. The fifth type, or koniocortex, is distributed in a thin band immediately posterior to the rolandic fissure, in the small area of Heschl's convolution in the temporal lobe, on either side of the calcarine fissure, and in the postsplenial and hippocampal region immediately adjacent to the agranular cortex just noted. The last two probably serve as afferent terminals for the impulses underlying smell and taste, and in the other three there are obvious terminals of impressions underlying touch, hearing and vision. The type II cortex is most spread out, covering large areas in the frontal, parietal and inferior temporal convolutions, as well as most of the mesial surface of the hemisphere. This type of cortex, it will be noted, is more closely related to the motor agranular cortex than to other types. The type III cortex, tending more toward the sensory form, occupies the inferior parietal and superior temporal area, the temporo-occipital regions and temporosphenoidal area, with an isolated, large patch close to the frontal pole. The type IV, or polar cortex, which may be only a modification of type III, occupies an area adjacent to the calcarine cortex and the anterior pole of the brain. The six layers found in each of these five types differ among themselves and allow one to define more than 100 different areas which probably underlie functions to some extent different. The time is far distant when the function of each of these fields will be known, but it would seem that the type I and type V are definitely established as motor and sensory for simple functions, that type II is more executive and type III more receptive, while type IV is not yet well understood. Man differs from lower animals as greatly in the extent of the intermediate types of cortex as in anything else. The lower animals have a cortex that is fully differentiated and mainly of two types, type I and type V. In man, one finds vast areas of more highly organized cortex which is yet less sharply differentiated into afferent and efferent characters.

The conclusion to be drawn from this study is that with greater development and individualization of the cortex new mental acquisitions are gained; in other words, progressive cerebration. But, since progressive cerebration implies not only an exaltation of existing faculties but also an augmentation of the mass of the brain, the formation of new cortical organs actually underlies new psychic faculties and, since there is no cause to believe that this natural law has ceased to function, one has the right to hope for still further development of the human brain.

FREEMAN, Washington, D. C.

THE ANATOMIC SUBSTRATUM OF HEMIBALLISM AND HEMICHOREA. EUGEN WENDEROWIC, Ztschr. f. d. ges. Neurol. u. Psychiat. **114**:78 (April) 1928.

Wenderowic reports a case of hemiballism in a woman, aged 81, who one week after the onset of her illness developed involuntary movements of the left side. Simultaneous with this she developed disorientation in space. These involuntary movements were in the nature of a hemiballism which persisted for one month until death ensued. They disappeared a few days before death, due, as Wenderowic believes, to the fact that the patient was comatose. At times the involuntary movements were more marked than at others, and periodically—about every two days—hemiballistic movements occurred in the extremities of the right side. On the basis of these clinical facts, Wenderowic states that the

basis of the symptoms was a thrombotic lesion in the regio subthalamica and that there was also one in the occipital lobe to explain the spatial disorientation. He says that it is hard to conceive of two thrombotic lesions occurring simultaneously in two regions so widely separated from one another. He believes that the thrombosis occurred first in the regio subthalamica and that this did not produce any symptoms until the thrombotic lesion in the cerebrum occurred, this acting as an inciting cause of the movements of hemiballism.

At necropsy, Wenderowic found an area of softening of one month's duration in the right hemisphere, involving the cephalad portion of the cuneus, the surrounding part of the calcarine lip, the cortex and white matter of the gyri concerned, and the medial portion of the forceps major. There was also a small area of softening in the region embracing the corpus subthalamicum, cerebral peduncle and internal capsule. In addition, there were a number of fresh miliary thrombi and hemorrhagic foci scattered throughout the basal ganglia, especially in the lateral nucleus of the thalamus, in the pallidum in both putamens, in the head of the caudate nucleus, in both internal capsules and in the claustrum. Wenderowic concludes that these foci are of no significance because they are so small. The brain stem, cerebellum and cord did not show anything of value.

A more careful survey of the lesion in the regio subthalamica revealed that the area of softening surrounded two thrombosed vessels and involved the corpus subthalamicum, the adjoining portion of the internal capsule (posterior limb) as well as the pes pedunculi. The corpus subthalamicum was most involved in its medial portions, the ventral and dorsal parts being but little affected. Wenderowic found that the destruction of the corpus subthalamicum involved only about one tenth of its normal substance. In the region of the posterior capsule the area of softening was about 1 cm. in diameter and involved field F *2* of Forel; the lateral portion of the pallidum was also destroyed by softening. The ansa lenticularis was not involved. Wenderowic calls particular attention to the destruction of Bonhoeffer's tract — the tractus dentatus cerebelli — thalamus contralateralis — regio gyrus centralica.

Before it is possible to determine the anatomic substratum of hemiballism it is necessary to determine what hemiballism is and whether it is an irritative or a destructive phenomenon. Kussmaul believed that hemiballism was a hyperkinesis sui generis, while Monakow stated that it was merely a form of hemichorea. Wenderowic says that clinically he would intuitively place it in a different class from chorea, since there are no absolutely hard and fast distinctions between the two. He believes, however, that hemiballism and hemichorea are identical, or are at least in the same class of disorders. Why, then, should a lesion in the regio subthalamica produce a special kind of chorea? Wenderowic believes that this is due to the fact that the chorea in this region is conditioned by a certain secondary apparatus which is situated in the subthalamic region. Is hemiballism an irritative, an Ausfall or a block phenomenon? Wenderowic believes that it is an irritative phenomenon. His reasons are: (1) The lesions causing hemiballism or hemichorea are all small and thus cause an insignificant destruction of the brain. It could not, therefore, be an Ausfall symptom. (2) Hemichorea is often accompanied by hemialgesia and hemiparesthesia, sensory disturbances which are unquestionably irritative phenomena referable to the thalamus. (3) In a case of Niketin and Wenderowic's in which the tractus dentatorubrothalamocorticalis was destroyed, involuntary symptoms did not occur during a period of more than a year, because there were no irritative manifestations of the systems involved. Destruction could not cause these symptoms. Wenderowic believes that hemichorea is an irritative phenomenon, that it is often due to a purely mechanical stimulation of Bonhoeffer's tract and that this is produced by fresh hemorrhagic extravasations, cysts of softening and tumors. On this basis it is easy to explain negative symptoms, because in the first place a lesion may be so placed as to be incapable of producing an irritation of Bonhoeffer's tract, and again this tract may be destroyed and thus there may not be a basis for the production of the irritative phenomena. In the production of hemichorea, Wenderowic rules out of considera-

tion, the pyramidal, frontocerebellar, cerebellocentral, pallidonigral, ansa lenticularis and other long and short tracts. The corpus subthalamicum plays only a secondary rôle in the production of hemichorea. In Wenderowic's case it was nine-tenths intact, while in the cases of Economo, Fiselen and Jakob it was entirely destroyed. In view of the latter observations, it is hardly conceivable that irritation of this ganglion plays much of a rôle in the production of hemichorea. If it does play a part, it is purely secondary. The chief rôle is played by the cerebellocerebral pathway, irritation of which causes hemichorea and hemiballism. In every case of hemiballism reported in the literature, there has been involvement, in one place or another, of the cerebellocerebral pathway. It is not necessary, therefore, to seek further for the pathogenesis of hemichoreiform movements. They are due to irritation of this tract which carries subconscious impulses of deep sensibility to the central motor area and there in turn, by direct or indirect connection, are in some way transformed into choreiform movements. The diffusion of these movements may be by means of the corpus callosum.

Wenderowic states that the cases of Liepmann-Vogt and Tromner-Jakob, on which depend the Ausfall theory of choreiform movements, really support his theory, because in both of these cases he was able to find a small lesion situated close to the cerebellocerebral pathway, irritation of which caused the choreiform movements.

ALPERS, Philadelphia.

STUDIES OF THE IRON REACTION IN THE BRAIN: I. INVESTIGATIONS IN NORMAL OR NONPARETIC BRAINS. E. VON ZALKA and T. VON LEHOCZKY, Arch. f. Psychiat. **85**:220 (Sept.) 1928.

The authors report the results of investigations of iron reactions in nonparetic brains. Within recent years it has been shown by Spatz that the brain tissue in paresis shows a characteristic form of iron reaction. It has been shown also, however, that in normal brains, especially in older persons, some parts of the brain give positive iron reactions. It was for this purpose that the authors investigated 100 brains of people who had not suffered with paresis in the following way: 1. A macroreaction was obtained by placing fresh brain tissue in a saturated aqueous solution of ammonium sulphide for at least two hours and then examining these for noticeable changes in color. 2. A microreaction was obtained by staining such tissues pressed onto a slide or tissues embedded in paraffin by the Turnbull method. Among the 100 patients examined, 14 died of carcinoma, 27 of sepsis, 12 of tuberculosis, 12 of arteriosclerosis (in 5 cases cerebral), 1 of congenital syphilis, 5 of syphilitic mesaortitis, 6 of lung disease, 4 of cirrhosis of the liver, 10 of vascular disease, 1 of meningeal tumor and 2 of intrameningeal hemorrhage. The ages of the patients ranged from 4 months to 80 years.

The results were as follows: The diffuse macroscopic reaction of a change in color was found mostly in the globus pallidus, the substantia nigra, the nucleus caudatus and, at times, the thalamus. It was less marked in children below 9 than in adults. In cases in which there were hemorrhages or areas of softening one could also see typical iron reactions about the lesions. The microscopic examination showed granular iron in fifty-six of the cases (seven times in the nucleus caudatus, thirty-two in the putamen, thirty-eight in the globus pallidus and twenty-three in the substantia nigra). In none of the cases was the granular iron found either in the thalamus or in the cortex. It is never found in children below the age of 13. It is rather rare between 13 and 25, and is most frequent in persons above 56. The iron granules were of different shapes and appearance, and seemed to be found most frequently in or about the glia cells and the connective tissue elements. In rare cases (mostly in the vicinity of a hemorrhage) they were found inside the nerve cells. Occasionally, free iron flakes were seen, apparently having no connection with any of the cellular elements.

MALAMUD, Foxborough, Mass.

A Study of the Presenile Psychoses. P. I. Lusternick, Collected Papers, Psychiatric Clinic of the First Moscow State University **2**:7, 1927.

The author reviews in detail the extensive literature concerning the subject of involutional melancholia, and states that as yet there is no definite and clearcut conception of this clinical syndrome. Twelve cases were studied in great detail, especially in reference to the prepsychotic personality. An investigation of these cases showed that three of the patients had a psychothymic personality, four were schizoid, one was psychoneurotic, one was a chronic invalid, one was paranoid, one epileptoid and one had genuine epilepsy. The clinical picture is largely colored by the prepsychotic personality of the patient, the constitution and the sensory endowment which lend a great deal of variation to the symptomatology in the cases.

Long before the appearance of the psychosis the author found a definite personality change, together with an impairment in general social efficiency. There is increasing difficulty in dealing with the ordinary problems of life, marked egoism, narrowing of the sphere of interests and a marked tendency to focus all the attention on one's self. There is also a certain loss of acuity in dealing with situations demanding fine discrimination and tact, although previously the patient was able to handle similar situations without any difficulty. Long before the onset of the psychosis, there is a tendency to harp on his own difficulty and marked vulnerability of the psyche of the patient to small insignificant insults. This is accompanied with a panic and a feeling of complete helplessness whenever complicated situations present themselves. There are constant uneasiness, restlessness and loss of self-confidence. The whole elastic fabric of social adjustment seems to be impaired. Some of these symptoms may be present to a mild degree in normal people past middle age. The author speaks of a triad which is found in the pathologic climacterium in both sexes. It consists in helplessness, egocentricity and narrowing of interests. The change from adult life to old age is essentially a normal physiologic process and is a normal biologic reaction. Like all other processes in the human organism, this change is occasionally accompanied by pathologic variations which express themselves in the involutional or the presenile psychoses. As to the influence of early arteriosclerosis or endocrine changes, it is too early in the light of present knowledge to make any dogmatic statements.

Kasanin, Boston.

The Malady of Pick: Circumscribed Senile Atrophy. Urechia and Mihalescu, Encéphale **23**:803 (Nov.) 1928.

Pick's disease has been previously described in fifteen women and nine men, ranging in age from 42 to 72 years, with more than one half of this number below the age of 60. This argues strongly against its consideration as a form of true senile dementia. The course varies from two to twelve years, usually from two to four. The etiology is obscure; it has been thought to be toxic or infectious, familial, related to epilepsy or psychopathy or other conditions. The symptoms are progressive, with dementia fairly marked, usually within a year. This is manifested by failure of memory and development of taciturn or indifferent attitudes: sometimes euphoria, acts of violence, impulsive sexual or exhibitionistic expressions, evidence of psychic infantilism and other manifestations. Hallucinations, delirium and confabulation are rare. Eventually the patients show extreme cachexia, but physical examination usually shows little else. The pupils, reflexes, blood pressure and spinal fluid are all normal. Pathologically, thickening of the skull, plaques in the meninges and absence of arteriosclerosis in the vessels of the base are usually present. The brain is striking in the amount of atrophy presented — often it weighs less than 1,000 Gm. This is often most pronounced in the frontal and adjacent regions, in the cornu ammonis, gyrus angularis and cingulum, but does not appear to follow definitely any well defined law, either of myelinization or of phylogenetic appearance. Microscopically, the lesions may be degenerative or inflammatory, with chromatolysis and eccentric position of the nucleus. Argentophil corpuscles are present in some of the cases; hypertrophy of the glia and amyloid deposits are common.

A new case is reported in detail.

Anderson, Kansas City, Mo.

THE REACTION OF THE OLIGODENDROGLIA TO INJURY OF THE BRAIN. ARMANDO FERRARO and LEO M. DAVIDOFF, Arch. Path. **6**:1030 (Dec.) 1928.

Experiments consisting of the aseptic removal of a portion of the cerebral cortex were performed on twenty-two cats. At intervals varying between twelve hours and sixty days after the operation, the animals were killed and the brains removed, and the region of the lesion was fixed immediately in Cajal's solution of formaldehyde-bromide mixture. Frozen sections were stained for oligodendroglia and microglia. The sections thus prepared were examined with a view toward understanding the rôle played by the oligodendroglia in the general reaction to brain injury. Special attention was paid to the question whether or not these cells participated in the formation of scavenger cells, and to the manner of this participation if it existed.

The following observations are quoted: The oligodendroglia cells were found to react to cerebral injury by acute swelling, atrophic degeneration, hypertrophy, hyperplasia and occasionally by the fusion of several cells into clusters. It was possible also to demonstrate the formation of oligodendroglia cells into compound granular corpuscles and to follow the various stages in this transformation. The change may occur directly from a normal cell or through an intermediary stage of swelling or hypertrophy.

The authors believe that the finding of a common origin for both astrocytes and cells of the third element, whether ectodermic or mesodermic, would probably do most to harmonize the apparently conflicting results of many investigations. They are apparently unaware of the excellent work of Harvey and Burr on this question.

WINKELMAN, Philadelphia.

THE PATHOLOGICAL ANATOMY OF CASES OF GENERAL PARESIS WITH CATATONIC SYMPTOMS. M. O. GURWITCH, Collected Papers, Psychiatric Clinic First Moscow State University **2**:225, 1927.

The catatonic syndrome in paresis has always attracted the attention of the psychiatrist. Bleuler and Rüdin explain it by a special constitutional predisposition of the patient, while Schröder and Häffner state that it is due to the special localization of the pathologic process.

The author describes in detail six cases with positive neurologic and serologic evidences of paresis. In spite of these observations the clinical picture resembled so closely the typical cases of catatonic dementia praecox that in some cases a diagnosis of dementia praecox was made by the clinicians. At autopsy, the gross microscopic examinations of the brain showed as a rule comparatively little involvement of the cortex; it usually consisted of a mild leptomeningitis. The most significant changes were found in the basal ganglia — striatum, thalamus and neostriatum. The changes consisted of marked increase in blood vessels, nerve cell degeneration, small hemorrhages, glia proliferation, small celled infiltration, etc. The picture was somewhat complicated by the fact that some of the patients were treated with malaria.

The author believes that the observation may be of some significance although he states that he has observed cases of paresis with marked involvement of the basal ganglia which, in course of the disease, did not display any catatonic symptoms. Further study should be made to see if similar observations will be found in typical cases of catatonic dementia praecox.

KASANIN, Boston.

TRAUMA AND TUMORS. LEILA CHARLTON KNOX, Arch. Path. **7**:274 (Feb.) 1929.

The important thing in this article for the neurologist is the question of trauma and tumors of the brain. The author states that even writers who did not admit the traumatic origin of bone tumors were inclined to acknowledge a probability that some of the cases of glioma reported were related to cerebral injuries. The literature on this is discussed in detail, and the conclusion is reached

that modern methods of histologic technic have shown that the gliomas form a complex group and that there is always danger of confusing certain types of proliferative gliosis with true neoplasms. The conclusion seems correct when the author states: "It is therefore the tendency of the more recent writers to question seriously a traumatic origin for glioma as for the other types of tumor and to feel that logically there is no reason to connect whatever injuries may have been sustained by the head years before with the presence of a primary tumor of the brain."

The general subject of what may be expected to be the effect of a trauma on a preexisting tumor is important, as, unquestionably, positive results will be presupposed in every case by the lay mind, and many of these cannot be scientifically excluded. The conclusion in this regard is that many of the recorded cases in which compensation has been awarded are instances of trauma merely calling attention to a preexisting tumor, either in the organs or in the connective tissues. In the mere presence of such a tumor a valid basis for compensation does not exist.

WINKELMAN, Philadelphia.

THE PINEAL BODY. EDITORIAL, J. A. M. A. **92**:561 (Feb. 16) 1929.

The pineal body has long attracted medical attention. The absence of exact knowledge regarding its function has caused an indulgence in the realm of fancy. A direct connection of this gland or body with the nervous system, as well as with the blood vessels of the choroid plexus, has been established. Horrax and Bailey have shown that its cells are specific.

Volkmann considers the pineal body to be incretory and to have a physiologic relationship to the ductless glands, especially to those having to do with sexual characters. On the other hand, Bauer believes that all the evidence is conflicting and that neuroregulatory rather than hormonal influences are involved in such conditions as the pubertas praecox syndrome in epidemic encephalitis. Walter also believes that the effects are neuroregulatory rather than that there is an internal secretion. Marburg and others summarize their views by the statement that the organ has a function in man and other mammals, and that this function is possibly an internal secretion which is not indispensable to life. Contrary to Berkley, Timme, Hoskins and Goodhart, however, they insist that there is no significant evidence that the secretion is of importance in the development of puberty.

Jelliffe's recent review of the literature from 1921 to 1927 shows that knowledge of the pineal body is fragmentary and that progress must depend on systematization, correlation and research. He considers that the preponderance of evidence is in favor of a neuroregulatory function rather than one of internal secretion.

CHAMBERS, Syracuse, N. Y.

THE ENDOCRINE TOXIC EPILEPSY. M. I. SEREISKY, Collected Papers, Psychiatric Clinic of the First Moscow State University **2**:185, 1927.

Epilepsy is often associated with disturbances of the glands of internal secretion. The thyroid gland especially has been mentioned often as being involved in the etiology of various convulsive disorders. The most common form of involvement of the thyroid in epilepsy is connected with hypothyroidism. In a fairly large series, a definite diminution in weight of the thyroid gland up to 20 Gm. has been observed. Hypothyroidism in connection with epilepsy is rare. Association of epilepsy with exophthalmic goiter or of the latter with epilepsy is infrequent. Epilepsy may appear before the onset of exophthalmic goiter, and at times hyperthyroidism precedes the onset of convulsions. The third manner in which hyperthyroidism and epilepsy are associated is when both these conditions appear approximately at the same time. It is in this group of cases that the author is especially interested.

The author reports four cases in which the onset of epilepsy and hyperthyroidism occurred at about the same time. It is interesting that in all these cases there was a history of epileptic personalities among the ancestors. Even before the onset

of epilepsy the patients were described as having been extremely egocentric and temperamental, having rigid personalities of the type usually described as "epileptoid." The author believes that there is probably some relation between the two and postulates a hypothesis that the same toxic factor which caused epilepsy also caused the appearance of the exophthalmic goiter.

KASANIN, Boston.

NARCOTIC ADDICTION. Current Comment, J. A. M. A. **92**:562 (Feb. 16) 1929.

A clinical study of addicts to narcotics was made in the wards of the Philadelphia General Hospital under the auspices of the Committee on Drug Addiction in affiliation with the Bureau of Social Hygiene, Inc., of New York; this was part of a comprehensive investigation, begun in 1921, of the biologic, medical, social, economic and legal aspects of narcotic addiction. The two purposes of this clinical study were: (1) to reveal any objective, physical, chemical or physiologic changes differentiating the addict from the normal person, and (2) to make a similar study of the addict when he is suffering from "withdrawal symptoms."

Light and Torrance recently published the first of a series of papers on this subject, dealing with the addict while he is still receiving the drug. The only differences noted were slightly lower hemoglobin content and red cell counts, a tendency to rather wide variations in the white cell counts and slightly high cholesterol and lactic acid contents in the blood serum. The study shows no physical deterioration nor impairment of physical fitness aside from the addiction itself. It is encouraging that if addiction can be relieved the addict may be rehabilitated.

The challenge is the need for further scientific research into the initiating and maintaining factors in addiction and into control methods for these factors.

CHAMBERS, Syracuse, N. Y.

STUDY OF A CASE OF HYSTERIA; ASSOCIATION OF HYSTERICAL TROUBLES WITH AN EXTRASYSTOLE ARRHYTHMIA. J. TINEL and G. MICHON, Encéphale **23**:891 (Dec.) 1928.

A woman, aged 32, of excellent physique, gave a history of symptomatic depression at the age of 10, and of attacks of prostration and difficult walking following an injury to the head at the age of 28; otherwise she had been well until February, 1928, when another emotional shock had precipitated a series of symptoms that were typically hysterical. There were pain, fainting, severe jerkings of the entire body, attacks of weakness, extreme fatigue, etc. At the hospital she was subjected to a severe reprimand, with almost instantaneous recovery. As the authors state, it would pass for a classic case of hysteria, produced by emotional shock and removed by a form of psychotherapy. However, this is not the whole story; she had simultaneous attacks of marked arrhythmia, extrasystolic in type, with tachycardia, tracings of which are presented. This is looked on as evidence of the real presence of an abnormal physiologic state. It was also found that attempts to elicit the patellar reflex often caused the onset of fainting attacks, and that, with this, graphic evidence of a marked peripheral vasoconstriction was obtained; in other words, underlying the entire syndrome was a disturbed autonomic balance.

ANDERSON, Kansas City, Mo.

THE AFTER-CARE OF POLIOMYELITIS. WALTER M. DICKIE, J. A. M. A. **91**: 19 (Nov. 10) 1928.

Although primarily of orthopedic interest, this article warrants abstracting. A brief description of the recently passed Crippled Children's Act is presented, and this is followed by a description of California's facilities for handling this problem. Tables are presented, showing age groups, dates of onset by weeks, distribution of paralysis and spinal fluid observations. In 1927, there were 1,274 reported cases of poliomyelitis; 812 showed paralysis in one or more muscle groups, 205 were abortive and 257 were of an undetermined type. A wider interest in serum therapy

was noticed: the patients in 247 cases were treated with convalescent or Rosenow's serum; 103 received the former with 65 improved, 25 showing no change, 24 showing aggravation and 19 having unknown results; 99 received the latter with 55 improved, 18 showing no change, 20 being aggravated and 6 having unknown results; 15 received both serums with 5 improved, 3 no change, 3 aggravated and 4 unknown. Symptomatic treatment is indicated early when there is an acute hemorrhagic myelitis and meningitis with a severe systemic infection. There should be complete function rest and no meddlesome intervention. A consideration of the etiology of deformities and a description of them and their treatment, the latter taken up by stages, is then presented.

CHAMBERS, Syracuse, N. Y.

THE CONDUCTION OF THE NERVOUS IMPULSE THROUGH THE PEDAL GANGLION OF MYTILUS. OSCAR W. RICHARDS, Biol. Bull. **56**:32 (Jan.) 1929.

The rate of nervous conduction in *Mytilus californianus*, a large Pacific coast mussel, was found to be 64.2 ± 1.8 cm. per second at temperature from 18 to 22 C., and that of *Mytilus edulis*, the smaller eastern mussel, was found to be 92.9 ± 3.3 cm. per second at 24 C. The rate of conduction through the pedal ganglion was much slower in both animals. Treatment of the ganglion by bathing it with strychnine abolished this delay. The application of camphor to the ganglion results in the foot exhibiting almost continuous movement with no apparent source of stimulation. These movements stop if the pedal ganglion is isolated by cutting the connectives just anterior to the ganglion. The longitudinal muscles of the foot are greatly relaxed and the circular muscles, especially at the base of the foot, contract when the foot is stimulated after the application of atropine to the ganglion. This is just the reverse of the response of the foot of the control animal to stimulation. These observations are discussed with respect to the effect of drugs on other animals and to the use of strychnine as a test for the function of the synapse.

COBB, Boston.

TRANSIENT INSULIN HYPOGLYCEMIC HEMIPLEGIAS. J. M. RAVID, Am. J. M. Sc. **175**:756 (June) 1928.

The case reported is of a diabetic patient in whom there were three transient episodes of complete hemiplegia, one lasting seven hours and two lasting three hours. Two of the three attacks of hemiplegia were typical of a lesion in the internal capsule; there was no residual paralysis. The patient is said to have been free from arteriosclerosis or cardiorenal disease, but he had hypoglycemia at the time of the attacks. The mechanism of the reaction is unknown; it is considered to be functional, since in other cases in which transient effects of insulin hypoglycemia have been noted it has been impossible to demonstrate any pathologic change in the central nervous system. However, it is suggested that in all insulin hypoglycemic reactions there is a definite involvement of the central or the peripheral nervous system. This action may be induced by the insulin itself or by intermediary products of carbohydrate metabolism and may manifest itself by alteration of function of the nerve elements themselves or of the blood vessels supplying them.

WAGGONER, Philadelphia.

HEMICHOREA DUE TO HEMORRHAGE INTO THE HYPOTHALAMUS. CHRISTOFREDO JAKOB, Arch. argent. de neurol. **2**:1 (Feb.) 1928.

Jakob reports a case, with necropsy, occurring in a man, aged 59, characterized by intense hemichorea, which began suddenly three days previously. Moderate mental confusion, with amnesia and disorientation, and uncertainty in walking were noted. The reflexes were about normal; muscular power was well retained. There were no major convulsions. During sleep the agitation disappeared. Alterations of vasomotor or secretory character were not observed. The condition persisted for twelve days when pneumonia supervened. The necropsy showed, in addition,

arteriosclerosis, with chronic myocarditis and cerebral arteriosclerosis. Section through the mammillary bodies disclosed a hemorrhage occupying the left corpus subthalamicum and spilling over slightly into the substantia nigra and base of the peduncle. However, in the serial sections there was a sharply circumscribed area of hemorrhage affecting the corpus subthalamicum and part of the zone incerta. The anatomy and physiology of this area are discussed.

FREEMAN, Washington, D. C.

AN UNUSUAL FORM OF HEREDITARY FAMILIAL DISEASE. M. SEREJSKI, Encéphale **23**:821 (Nov.) 1928.

The cases of four siblings, three men and a girl, aged 28, 22, 20 and 19 years, respectively, are reported with a symptom complex as follows: auditory disturbances without involvement of the vestibular portion of the ear or nerve, speech difficulties, retinitis pigmentosa, hemeralopia, dermographism, acrocyanosis and some degree of involvement of mentality. Tendon reflexes were exaggerated; there was evidence of hyperpituitarism — marked development of the lower jaw, hypertrichosis, increased metabolism and absence of sexual desire. The onset was insidious and occurred during childhood. Three other members of the same family were normal, as were the parents; five children died in infancy of infectious diseases. Syphilis could fairly be excluded. The author considers the condition a form of familial disease not definitely classifiable with any existing type, although related to amaurotic family idiocy.

ANDERSON, Kansas City, Mo.

BRIGHTNESS VISION IN THE DEER-MOUSE, PEROMYSCUS MANICULATUS GRACILIS. PAUL AMOS MOODY, J. Exper. Zool. **52**:367 (Feb. 5) 1929.

Deer-mice are largely nocturnal in habit. Since colors are not perceived as such in dim light by the human eye, the ability of the eyes of deer-mice to distinguish different intensities of light, rather than different wave lengths, was investigated. The apparatus and technic employed are described. Intensive training in discrimination between two white lights differing in intensity was given to six deer-mice, *Peromyscus maniculatus gracilis* LeConte. Six thresholds of discrimination of light intensity were found. The results were found to bear relationship to the Weber-Fechner law. The morphology of the eye and the retinal histology of the deer-mouse were studied. The visual-cell layer lacked "cones," whereas the "rods" were found highly developed. This is perhaps correlated with acuity of vision for brightness.

WYMAN, Boston.

THE EFFECT OF ELECTRICITY ON THE EYE: SOME MEDICOLEGAL CONSIDERATIONS. J. N. ROY, Ann. d'ocul. **165**:433 (June) 1928.

Roy concludes that the effect of electricity is not always grave; on the contrary, it is usually benign, and the inflammation of the eyelid and of the anterior segment of the eye, caused by the ultraviolet and the infra-red rays, frequently produces alarming symptoms. These symptoms, however, disappear in a few days. Lesions of the retina and optic nerve are rarely produced. Roy states that in one case he was positive that atrophy of the right optic nerve was caused by the electricity to which the patient was exposed. He thought the trouble had been produced by the visible radiation from the metal which had become incandescent from the electricity. The eye had been exposed for eight minutes at a distance of 130 cm., without protecting lenses.

BERENS, New York.

Society Transactions

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Dec. 20, 1928

LOYAL DAVIS, M.D., *President, in the Chair*

TUMOR OF THE SEPTUM LUCIDUM AND CORPUS CALLOSUM CAUSING APRAXIA. DR. PERCIVAL BAILEY.

I have thought this case of interest because of the unusual location and symptomatology. The clinical record is as follows:

R. H. A., aged 57, a business man, was admitted to the Albert Merritt Billings Hospital on Nov. 2, 1928. He had suffered from stomach trouble for several years. After a gastro-enterostomy in 1918, the patient was in good health until August, 1928, when he had an attack of vomiting and headache which lasted for only a few hours. He continued in business, but in September it was noticed that he was lethargic and sleepy. He lost his memory for business details and became indifferent to his work. He was taken to Battle Creek Sanatorium. While there he was confused, indifferent and disoriented, had several attacks of projectile vomiting and complained of frontal headache. No evidence of focal lesion of the nervous system was found; there was no choking of the optic disks. The spinal fluid contained 5 cells per cubic millimeter; sugar, 82 mg. per cubic centimeter; Pandy test, 4 plus; Wassermann test, negative, and gold chloride, 34555440000.

The patient was transferred to the North Shore Health Resort on October 14, under the care of Dr. William G. Stearns. At this time he was dull and somnolent and vomited occasionally, but had little headache. On October 29, while walking with his wife, he reeled suddenly and would have fallen but for support. Subsequently, he vomited and seemed worse. His pulse was weak, and he perspired freely. On November 2, he was seen by Dr. Peter Bassoe, who referred him to the Billings Hospital.

On admission, the pulse rate was 140 and the temperature 103 F. The patient did not speak; his eyes were turned to the left, and he breathed rapidly. There was a spontaneous nystagmus to the left, and a slight weakness of the left side of the face. An examination disclosed normal superficial and tendon reflexes. Reaction to pin-prick was prompt over the whole surface of the body. There was a low grade choking in each eye, 2.5 diopters in the right and 1 diopter in the left, with edema, fibrin and hemorrhages in the region of the disks. Dr. Kronfeld thought that the secondary changes in the retinas were too great to be accounted for by the degree of choking.

The outstanding phenomena, however, were a myoclonic twitching of the entire musculature, involving also the diaphragm, and a total apraxia. The twitchings were exactly of the type seen in acute encephalitis. These twitchings, together with the fever, sleepiness, stiff neck, and positive Kernig and Brudzinski signs, seemed to indicate the presence of encephalitis until the roentgen examination disclosed the fact that the dorsum sellae appeared to be completely absent.

The apraxia was complete. When any object was placed in the patient's hands he grasped it with great force, proving the absence of paralysis. He did not experience any difficulty when he moved his arms to perform involuntary movements, such as scratching his head, but was totally unable to use them to any purpose. The same was true of the cranial musculature. He made serious attempts to talk and gave every indication of great distress and sometimes anger but only once succeeded in forming an intelligent phrase. When food was placed in his mouth he rolled it around and made queer contortions of the facial and

bodily musculature, but swallowed only if the food was placed far back in the pharynx. Water was almost invariably spilled from the mouth without being swallowed. The patient never choked when swallowing, however, and after a little experience it was possible to feed him satisfactorily by placing soft food far back in the pharynx.

It was difficult to form an accurate opinion of the mental condition. The patient recognized his wife and would indicate approval and pleasure when she happened to realize what he was trying to say. He sometimes indicated approval of simple questions. It was impossible to test him for comprehension by commands which he was totally incapable of carrying out.

He had periods of tachycardia, but an electrocardiogram could not be taken during these periods. Nothing abnormal was found in the lungs at this time.

A diagnosis was made of tumor of the base of the brain, involving the corpus callosum. Since a bad prognosis was given, confirmatory evidence was sought in ventriculography. On November 5, the right lateral ventricle was punctured. About 20 cc. of dark bloody fluid escaped, mingled with clots; 15 cc. of air was injected. The roentgenogram showed a dilated posterior and temporal horn, a

Fig. 1.—A tumor arising from the septum lucidum and destroying the anterior portion of the corpus callosum.

cystic cavity, about 3 cm. in diameter, in the right frontal region and some bubbles of air in the third ventricle. On November 6 the left ventricle was punctured; 20 cc. of clear fluid was removed, under no tension. The same amount of air was injected. The roentgenogram showed a dilated posterior and temporal horn on the left side also, with no air in the frontal region. Following these punctures the patient was better for a few hours, but on November 7 he began to have Cheyne-Stokes respiration and the choking of the optic disks had increased. A right subtemporal decompression was made. There was considerable subarachnoid fluid, but to the operator's surprise, the brain was under little tension. The patient improved somewhat and was given roentgen treatment in the right and left temporal regions on November 9. On November 10, he died.

Necropsy was performed four hours post mortem and disclosed the following facts: nodular hyperplasia of the left and middle lobes of the prostate gland, thrombosis of the hemorrhoidal and periprostatic veins, fibroma of the spleen, diffuse fatty infiltration and degeneration of the liver, moderate arteriosclerotic changes and glomerulonephritis of the kidneys, moderate fatty changes in the aorta and coronary arteries, acute distention of the right side of the heart, bilateral adhesive fibrous pleuritis, acute hypostatic hyperemia and bronchopneumonia of

both lungs, healed calcareous and fibrous tuberculosis of both lungs, a healed posterior gastro-enterostomy wound, fresh operative wounds in the cranium, and a tumor of the brain.

An injection of solution of formaldehyde was made into the brain through the carotid arteries. On removal, the convolutions were seen to be greatly flattened and smooth over the frontal lobes, but fairly normal in appearance over the temporal, parietal and occipital regions. The orbital surfaces of the frontal lobes bulged downward and backward. The optic nerves were much flattened, and the hypophysial stalk was firmly adherent to the under surface of the bulging infundibular region. The hypophysis was flattened, and the dorsum sellae completely absent. The vessels at the base showed no evidence of arteriosclerosis.

The medial sagittal section of the brain disclosed a tumor involving the corpus callosum in its anterior third and the septum lucidum (fig. 1). The third ventricle was pushed backward. The right foramen of Monro was blocked by a clot of blood, and there were several small clots in the third ventricle. Parasagittal sections showed that the tumor extended laterally into both frontal lobes. The anterior horn of the left lateral ventricle was completely occluded. The anterior horn of the right lateral ventricle was reduced to a narrow slit which opened anteriorly into a necrotic cavity, 3.5 cm. in diameter. The tumor was necrotic and infiltrated by blood.

Microscopic examination showed the tumor to be of the type known as a protoplasmic astrocytoma, with large areas of necrosis and hemorrhage. A few mitotic figures and a few multinucleated cells were seen, so that the tumor was undoubtedly growing actively; most of the symptomatology, however, was probably due to repeated hemorrhages in the growth.

Comment.—I have never seen so definite an example of tension confined predominantly to the frontal region. Before the brain was fixed, the difference between the appearance of the frontal and occipital regions was striking. The absence of any great general intracranial tension, noted both at ventricular puncture and at decompression, is thereby explained, as is also the appearance of the optic disks.

The confusion, disorientation and loss of memory were probably attributable to extension of the growth into the frontal lobes, but the myoclonic twitchings are not so readily explained. Since they came on acutely following a sudden upset, I am rather inclined to attribute them to the intraventricular hemorrhage which the patient must have experienced, as indicated by the puncture of the right lateral ventricle.

The apraxia can scarcely have been due to anything other than the involvement of the corpus callosum, which was completely destroyed in its anterior portion. Theoretically, a lesion involving only the corpus callosum should cause a unilateral motor apraxia (Bailey: ARCH. NEUROL. & PSYCHIAT. **11**:501 [May] 1924). In the present case, the extension into the frontal lobes probably explains the bilateral apraxia which was doubtless of ideomotor type.

PAROXYSMAL CEREBELLAR DYSFUNCTION. DR. ROY R. GRINKER.

A white man, aged 28, entered the University of Chicago Clinics complaining of attacks of "drunkenness without alcohol." At the age of 12, without previous febrile disturbance or trauma, the first attack of dizziness occurred. At first these attacks occurred about once in six months, but lately they had increased in frequency and severity, maintaining, however, the same general characteristics. They now appear about once a month, although as many as three a week have occurred. During the war, however, while the patient was in the navy for thirteen months he had no attacks.

The attacks begin at any time of the day but especially after overexertion or when the patient is fatigued or excited. An artificial fright, which was staged seriously and in which he was accused of stealing while in the hospital, failed to produce an attack, whereas an argument with another patient produced one. The attacks increase in severity with a sort of crescendo effect, often taking several hours before the maximum dysfunction appears.

The patient staggers like a drunken man and many times has been accused of drinking. He has even fallen off street car steps. His arms become ataxic so that he is unable to write, convey food properly to his mouth or do any work. His occupation is that of motorman. During one attack, he continued to drive his street car for six hours, stopping it each time with a terrific jolt. His speech is slurred and difficult to understand, and his tongue seems thick. In severe attacks, he vomits and is rather drowsy. Consciousness is never lost. Relief from the subjective sensations is obtained by lying down. Curiously, he becomes normal only after he closes his eyes and actually sleeps for ten or fifteen minutes.

The patient is an only child with a family history that is without significance. He had measles, mumps, chickenpox and scarlet fever as a child and severe grip for two weeks at the age of 15, three years after the onset of the present trouble. He has been married twice, having a normal child by each wife.

Examination of the patient in an interval between spells revealed nothing abnormal either generally or neurologically.

During an attack, the patient has a pulse rate of 110 and the heart tones are accentuated, but the blood pressure is still 130 systolic and 80 diastolic. There is no nystagmus. The speech is slurred and thick and resembles a cerebellar speech in its frequent pauses for breath-taking, senseless stops and ebbs in volume. There is marked clumsiness and ataxia of the upper extremities with adiadokokinesis. Intention tremor is not present. The gait is ataxic; there is no tendency to fall in one direction. The Romberg sign is positive. The deep reflexes are more lively during an attack, and the abdominal reflexes are quickly tired.

The blood chemistry, including the carbon dioxide content, was completely normal. Wassermann tests of the blood and spinal fluid were negative. A faint trace of globulin was noted in the spinal fluid. A normal Lange reaction was obtained. A reading of the spinal fluid pressure was within normal range.

No evidence of labyrinthine disturbance was found. Bárány tests gave negative results and did not produce an attack. Douching the left ear with 90 cc. of warm water produced the same sensation felt during an attack and nystagmus to the right for forty seconds with marked past pointing to the left on both sides. The patient became nauseated and vomited. Douching the right ear did not produce the negative effect.

There can be no doubt that, at the present time at least, in the interim between attacks the patient is entirely normal and that during attacks he shows objective evidence of cerebellar disturbance only, without nystagmus and with preponderating loss of equilibrium, although ataxia, especially of the speech is present. It is interesting to observe here that the normal relation between respiratory movement and speech is upset, as Hiller has shown occurs so typically in Friedreich's ataxia and which he considers is a paleocerebellar disturbance.

Where should this condition be placed concerning its local origin? The frontal lobes should certainly be considered, since lesions here may well produce cerebellar effects. An organic lesion, however, must necessarily be bilateral to reside in the frontal lobes and produce bilateral cerebellar symptoms. The possibility of a frontal epilepsy may be mentioned, but the limitation of the discharge in such a small area for so long a time without the involvement of consciousness or psychic functions is unlikely, as is the possibility of a frontal discharge producing loss of selective cerebellar functions.

The possibility of an epilepsy limited to the cerebellum, its discharge arising there and becoming spent before it may proceed further, is interesting. If so, this case should be valuable in helping one ascertain the cerebellar component in the usual cerebral fit. The crescendo increase in severity and cessation of the attack after sleep should indicate an exhaustion rather than an excitation or discharge as one now conceives epilepsy; nor could a vascular spasm explain such diffuse cerebellar dysfunction without nearby symptoms.

One explains such conditions as Friedreich's ataxia by abiotrophy and all that this term implies. Can one not conceive of a periodic exhaustion of a certain

system or group of closely allied systems from which recovery may take place, but eventually leading to morphologic change? This suggestion seems to me worthy of consideration.

One seems to be dealing with a functional condition; by that I do not mean neurotic, but one as yet not associated with demonstrable anatomic manifestations. It is not, certain, however, that a tumor is ruled out, for such analogies as symptoms of narcolepsy in tumors of the third ventricle, as described by Percival Bailey, closely resemble this case in their periodicity. A tumor of the midline in the ventricular surface may be considered in this connection.

The similarity of the condition in this man to an acute alcoholic intoxication is striking in that volitional movements only bring out his cerebellar disturbance and subjective sensations. This coincides with Walsh's conception of the cerebellum as an aid or adjunct to the cerebrum in steadying and limiting voluntary, cortically originated movements.

DISCUSSION

DR. GEORGE W. HALL: How long has the patient had these attacks?

DR. ROY R. GRINKER: Since the age of 12. There is no history of similar attacks in other members of the family. Three years after the onset of the attacks, he had a severe attack of grip.

DR. GEORGE W. HALL: Are the attacks similar to those due to methyl chloride?

DR. ROY R. GRINKER: I am not familiar with the effect of methyl chloride. The principal feature of these attacks is that they are relieved by sleeping. Lying down will not relieve them; the patient must sleep, if only for a few minutes.

DR. PERCIVAL BAILEY: I saw this man in an attack, and he then had all the evidences of an advanced cerebellar lesion, with the exception of nystagmus. The speech was as typical of cerebellar speech as any that I have heard.

THE PATTERN OF SENSORY RECOVERY IN LESIONS OF THE PERIPHERAL NERVES. DR. LEWIS J. POLLOCK.

Only scanty references, often incomplete and faulty to the pattern of sensory recovery, may be found in the literature dealing with injuries of the peripheral nerves. When the sensory charts of complete lesions were compared with those of healing lesions, some interesting observations were made which merit description. For many years it has been noted that the total loss of sensation is limited to a much smaller area than one would expect from anatomic distribution. It has been well recognized that the residual sensibility of a nerve is more extensive than its accepted anatomic distribution.

It has been noted that about fifty days following the suture of a nerve the area of analgesia begins to shrink. This early return of sensibility to pain has been attributed to an early regeneration of protopathic fibers. In some previous publications, I stated my opinion that this early and dissociated return of sensibility to pain is due to the assumption of function by adjacent uninjured nerves.

The return of sensibility to pain in such cases follows a pattern so characteristic that it may be recognized at sight. It never returns in the distal phalanges of the little finger in lesions of the ulnar nerve nor in the distal phalanges of the index and middle fingers in lesions of the median nerve and so on. It always occurs along the borders of an uninjured nerve, and may well be described as a shrinkage.

The material that I have used consisted of about 400 cases. The general conclusions are derived from the whole material. A number of cases were selected in which recovery occurred after an operation contrasted with cases of spontaneous recovery. No difference could be detected between the cases in which recovery occurred following neurolysis, resection and suture, except in those in which the sensory loss did not occupy completely the sensory cutaneous distribution of the nerve prior to the neurolysis. Early return of function was found in the partly anesthetic or analgesic area.

The characteristic features of the sensory loss of regenerating nerves may be enumerated as follows: Return of sensibility to pain, touch or temperature in that area of the sensory distribution of a nerve which is supplied by it alone, the isolated sensory area of the nerve; return of sensibility to pain, touch or temperature in patches some distance from the area supplied by an adjacent uninjured nerve; return of sensibility to pain, touch or temperature in deep indentations; diminution of degree of loss of sensation of pain, touch or temperature in the isolated sensory supply of a nerve; return of sensation of pain, touch or temperature in the border between the sensory supply of two nerves simultaneously injured, and interlacing of the border of sensory loss of one type of sensation with that of another.

DISCUSSION

DR. S. W. RANSON: I am much interested in Dr. Pollock's work on the recovery of sensation after lesions of the peripheral nerves, because of its bearing on the question of the existence of different types of sensory nerve fibers. Head attributed the disassociation of sensation in lesions of the peripheral nerves to the existence in the superficial nerves of two distinct types of sensory fibers, the protopathic and epicritic. His interpretation has not met with general acceptance. I wonder what interpretation Dr. Pollock puts on this disassociation; whether it is a matter of the time required for regeneration of the peripheral end organs; whether, as Dr. Head thought, it represents different types of fibers, or whether it is a matter of reeducation in the central nervous system. Pain, having possibly more primitive and less specialized connections in the central nervous system, might return more rapidly than the highly specialized nerve connections.

One thing in favor of Head's interpretation is that there is undoubtedly a separation of the peripheral sensory nerve fibers into two types. The microscope shows the distinction between small fibers, most of which are unmyelinated, and large fibers, which are myelinated. Physiologic work indicates that pain is carried by the small fibers.

DR. PERCIVAL BAILEY: How does Dr. Pollock explain the recovery of function in an isolated patch in the center of the anesthetic area? What is the basis for the idea that each bundle of fibers has an isolated area of distribution?

DR. LEWIS J. POLLOCK: No conclusions can be drawn from the clinical studies that would have physiologic merit, beyond the one contention which I have held from the beginning. Dr. Head has contended that because of two things the validity of his theory stands: (1) the ease with which protopathic fibers regenerate; and (2) the first return of the sensation of pain, fifty days after his suture of the nerve, one of his strongest arguments. This, I think, is entirely fallacious. It was brought out by Sherren that in mild lesions, when pain returns, sensation to touch returns at the same time. In the cases reported, when sensation to pain returned before sensation to touch, it was always in the area of overlap, never in that of the isolated supply of the nerve, as the tip of the little finger in the lesions of the ulnar nerve. It was found that when there was a return of sensation in the isolated nerve supply, sensations to touch and pain usually began to return at the same time. At times, sensation to touch and not to pain would return, or sensation to pain might return and not to touch. Head said that he found a triangular zone in which sensation to touch had returned and that to pain had not. This proved to him that there are two separate pathways—an accurate observation. How one can interpret it I do not know. There is some validity to the thought that it may be a matter of education. It is interesting to know that there is never an overlap for sensation to touch, but always for sensation to heat, cold and pain. It is not known whether overlap occurs from a new growth of a hitherto uninjured nerve or whether there are hitherto undiscovered fibers already existing in the area.

I do not know how one would explain the return of sensation in islands. I may say, as Dr. Bailey knows, that there is considerable argument concerning the existence of fascicular pathways of peripheral nerves. Dustin pointed out that

there are numerous intraneural plexuses about the point of emergence in a fiber and that after a nerve fiber is given off, long pathways are reassembled. They may reassemble so that a certain group of fibers more often occupies one particular part of a cross-section of a nerve at a certain level. I do not believe that we have any internal topography such as Stoffel pointed out.

GUMMA OF THE THORACIC SPINAL DURA MATER. DR. RICHARD A. LIFVENDAHL.

A large gumma of the thoracic spinal cord arising from the dura mater was presented. The mass was 4 cm. long and 7 mm. in its greatest diameter. It was

Fig. 2.—Gumma of the thoracic spinal cord arising from the dura mater. *A*, cross-section.

located on the posterior and lateral aspects of the cord and caused a slight compression of the spinal cord at the level of the fourth thoracic vertebra.

The specimen was obtained from a colored woman, aged 51, with a four plus Wassermann reaction of the blood, who suddenly developed paraplegia. The spinal fluid, obtained by the lumbar route, was clear and yellowish and contained 450 lymphocytes per cubic millimeter; compression of the jugular veins resulted in no increase in the spinal fluid pressure. The general condition of the patient, as revealed by autopsy, was too far advanced to have warranted surgical intervention, in that acute cystitis with pyelonephritis and numerous pyelonephritic abscesses in both kidneys; also a gumma in the liver, and typical syphilitic changes in the thoracic aorta were found.

Histologic examination at the level of the tumor mass revealed it to be composed of caseous necrotic tissue with small amounts of connective tissue. This formed a fusiform swelling arising from the dura mater. The periphery was the site of chronic inflammatory changes consisting of endophlebitis, arteritis and perivascular accumulations of lymphocytes and plasma cells. The spinal cord at this level showed irregular and varying sized areas of degeneration which were most severe and extensive in the posterior and lateral columns of the right side but were present also in the left pyramidal tract and in the tip of the anterior column near the median fissure. These changes consisted of proliferation of the glia with numerous screen cells, many corpora amylacea, marked edema, diminution of the myelin sheaths, the lipoids taken up by ameboid glia cells and small areas of recent hemorrhage. The vessels in numerous areas were surrounded by lymphocytes. In the large motor ganglion cells there was chromatolysis, karyolysis and granular degeneration of the cytoplasm. Levaditi stains showed no spirochetes.

This type of syphilitic involvement of the spinal dura mater must be differentiated from the diffuse form, which is relatively more frequent. On microscopic examination, the structures above and below the level of the tumor mass were free from any syphilitic changes. From the surgical standpoint, the indented and roughly lobulated surface of the growth was considered in differentiating it from other tumors, particularly tuberculomas, which, as emphasized by Virchow, have a smooth and rounded surface.

DISCUSSION

DR. RICHARD H. JAFFE: Considering diseases from a historic standpoint, one sometimes finds that their character has changed. When one visits the medical museums one may see specimens of conditions once considered common which now are rare. On the other hand, one may miss specimens of diseases which now are observed frequently. Syphilitic aortitis, for example, one of the most common diseases of the cardiovascular system in the present day, was hardly known several decades ago. Old anatomic collections are crowded with gummas in various organs, while now they are rare.

There are two forms of syphilitic lesions of the dura mater: a diffuse pachymeningitis and the circumscribed gumma. The former condition, which does not cause a destruction of the dura mater, is frequent, while a true gumma which destroys the dura is rare. The histologic observations prove beyond doubt that in Dr. Lifvendahl's case one is dealing with a gumma. The endophlebitis, especially, is most striking. Spirochetes could not be demonstrated, the lesion apparently being too old.

DR. G. B. HASSIN: Did Dr. Lifvendahl study the condition of the spinal cord above and below the tumor and ascertain whether the changes were at the level of the tumor only? This, I think, is important. My assumption is that the tumor should have caused blocking of the circulation of the tissue fluids of the cord only at the level occupied by it, leaving the areas above and below intact.

DR. PERCIVAL BAILEY: I have never seen a gumma of the spinal cord, but have seen several gummas of the brain. Dr. Lifvendahl spoke of a gumma of the frontal lobe. I should like to ask him about that. I have been interested in differentiating between tubercle and gumma of the brain. If a tubercle is found at operation it should be let alone, but if the lesion is a gumma it is best to remove it. One differentiating point that I know is that a tubercle is not likely to involve the meninges, while a gumma is likely to involve the meninges first and the brain secondarily.

Concerning the treatment for a gumma involving the brain, there is a reason for not giving antisyphilitic treatment: Gummas in this region are notoriously resistant to treatment, but after such a lesion has been operated on, although nothing is done but to disclose the lesion and leave conditions as they were before, the lesion is favorably influenced by antisyphilitic therapy afterward. This observation has been made by several physicians, as I have noted from reading the recent literature on this subject.

DR. R. A. LIFVENDAHL: Replying to Dr. Hassin, the cord was studied above and below the lesion but no changes were found.

In reply to Dr. Bailey, the gumma of the right frontal region was not large, it was located in the frontal lobe and did not extend to the surface.

Last year, Dr. Grinker and I reported twenty-one cases of tuberculoma that had been observed at the Cook County Hospital during the last five years. We noted that near the edge the surface of the tumor was rounded, as compared to this gumma. Virchow stated that this is one of the differentiating points between tuberculoma and gumma.

REMISSION OF TABETIC SYMPTOMS FOLLOWING SACRAL INJECTION OF PHYSIOLOGIC SOLUTION. DR. ALBERT B. YUDELSON and DR. JOHN VERMEREN.

This article will be published elsewhere.

Regular Meeting, Jan. 17, 1929

LOYAL DAVIS, M.D., *President, in the Chair*

PRIMARY LYMPHOSARCOMA OF THE INTESTINES, WITH METASTASIS TO THE BRAIN. DR. PETER BASSOE.

I am indebted to Dr. Samuel J. Lang of Evanston for the privilege of seeing this interesting patient. A previously healthy man, aged 30, fell in a garage on Oct. 10, 1928, apparently on his hand; he almost immediately developed severe continuous pain in the right frontal region and came to the hospital on the next day. Morphine was required to control the pain. On October 15, the optic disks were pronounced normal by an ophthalmologist. Roentgenograms of the head revealed nothing abnormal. When I saw the patient on October 24, the disks were hyperemic and slightly blurred, and there was a slight Kernig sign but no stiffness of the neck. There was a suggestion of slight weakness of the lower part of the left side of the face, but no other paralysis. Sensation was normal. The left knee reflex was slightly stronger than the right; the other reflexes were normal. Lumbar puncture, on October 25, yielded a clear fluid with a cell count of 5. On October 27, the patient had a profuse intestinal hemorrhage, followed by stiffness of the neck. For three days the temperature was from 101 to 101.8 F.; then it gradually declined to normal. He was irrational much of the time. When I saw him again on November 10, he had bilateral papillitis, marked retractio of the neck, slight Kernig sign, slow nystagmus on looking to the left, deviation of the tongue to the left, incoordination of the left arm and leg, slight increase of the left knee reflex and normal plantar and abdominal reflexes. On November 11, Dr. Loyal E. Davis found weakness of the right external rectus muscle and marked nystagmus with the quick component to the side of fixation. The mental confusion and the retraction of the head persisted. There was no more hemorrhage from the bowel. The patient died on December 4.

Necropsy was performed by Dr. Paul S. Rhoads. There were three round, flat nodules, from 1.5 to 2.5 cm. in diameter, in the wall of the jejunum. They were raised and located chiefly in the submucosa. One was ulcerated.

On the undersurface of the right temporal lobe of the brain was a dark red globular mass, 4 cm. in diameter, which lay beneath the dura and caused a depression 3 cm. deep in the temporal lobe. The growth extended to the posterior border of the lobe so as to press against the tentorium, and reached within 2.5 cm. of the anterior pole. On section, this mass appeared to be made up chiefly of hematoma, but there was a flattened tumor growth along the dural surface. At the junction of the mesial and inferior surfaces of the right occipital lobe was another nodule, 2.5 cm. in external diameter, extending 1.5 cm. into the lobe so as to reach within 0.5 cm. of the posterior horn. It included part of the calcarine area. The whole upper surface of the cerebellum was dark and elevated, and on section a globular, partly grayish-white, partly hemorrhagic mass, 4 cm. in diameter,

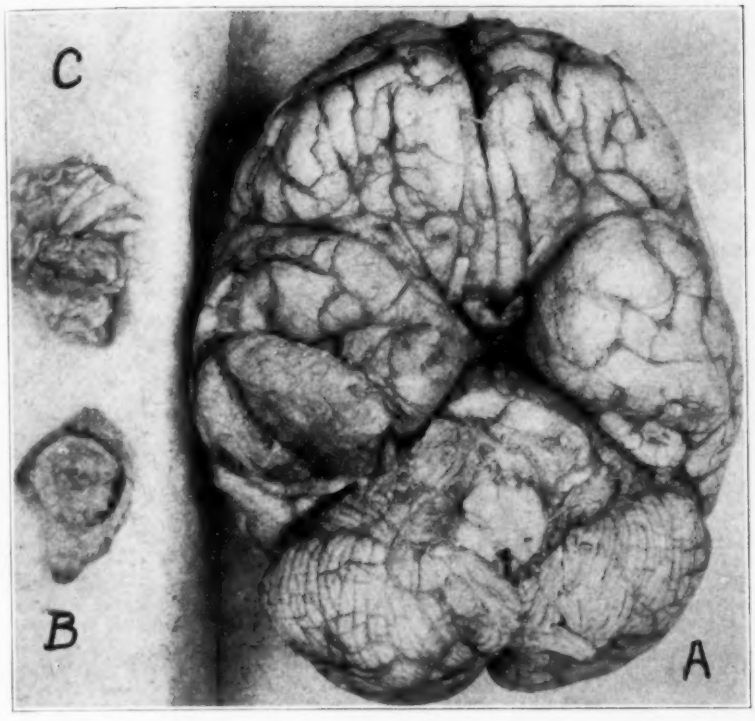

Fig. 1.—*A*, undersurface of brain showing tumor and hematoma in the right temporal lobe; *B*, ulcerated tumor of the intestine; *C*, another tumor of the intestine.

Fig. 2.—Tumor in the cerebellum.

was found to extend downward to the ependyma, which, however, was not penetrated. It was noted that this tumor was separated from that in the occipital lobe only by thick tentorium. Macroscopically, no tumor could be seen in the tentorium. Histologic examination showed that the intestinal tumor and those of the brain had the same structure, which was that of an extremely vascular round cell sarcoma. In one of the intestinal tumors, which were located in the submucosa, a vein was found filled with tumor tissue, which explained why metastasis to the brain occurred. Prof. E. R. LeCount examined the specimens and expressed the opinion that the tumor was a primary lymphosarcoma of the small intestine with metastatic tumors in the brain.

DISCUSSION

DR. LOYAL DAVIS: When I saw this man, his abdomen was extremely tender and rigid, so it seemed to me that there was no doubt that some abdominal pathologic changes were present. He also had symptoms which certainly pointed to a lesion in the posterior fossa, and I thought that because of the prominence of those symptoms, together with those which were due to a supratentorial lesion, little could be done surgically to expose all of the lesions, and that therefore surgical intervention was not advisable. I am greatly interested to see and hear the pathologic observations.

DR. EUGENE F. TRAUT: Is the color due entirely to hemorrhage?

DR. PETER BASSOE: Yes.

DR. ALBERT B. YUDELSON: Was there any history that gastro-intestinal disturbance had preceded this condition?

DR. PETER BASSOE: None.

BACILLUS FECALIS ALKALIGENES MENINGITIS, WITH RECOVERY: PRESENTATION OF PATIENT. DR. L. C. GATEWOOD.

M. K., a boy, aged 13, entered the Presbyterian Hospital on June 18, 1928; his chief complaints were recurrent spasmodic contractions of the left side of the face, arm and leg, with headaches and dizziness, occasionally followed by salivation and unconsciousness. When 1½ years of age, the patient fell from a porch, receiving a severe blow on the head. He was in a hospital for one week, and his physician reported that he had a fracture of the skull. Subsequently, a large, hard bump developed. Except for an attack of rheumatism at the age of 6, the boy had been in good health. At the age of 10, he began to have headaches and, later, occasional twitching of the left side of the body. These would last for two or three minutes and would occur two or three times a month. About two years before presentation, a period of freedom from attacks occurred, lasting six months. For the eighteen months before presentation, the attacks had increased in frequency and severity so that on admission to the hospital he was having several a week. A typical attack began with contractions of the muscles in the left side of the face, which drew the corner of the mouth outward. The left arm and finally the left leg would then become rigid. The average attack lasted about three minutes and, according to the mother, the patient was conscious during most of them. After an attack, he complained of general weakness and usually slept for from fifteen to thirty minutes. A dull aching pain over the right eye would persist for from fifteen to twenty hours. The boy's mother knew of no definite exciting cause for the attacks. Dizziness usually followed the attacks and occasionally nausea and vomiting. Strabismus had been present as far back as the mother could remember. The history was otherwise unimportant except for anesthesia of the right index and middle fingers following a lacerated wound of the wrist which occurred in April, 1928. The family history was negative for insanity or epilepsy.

On examination, there was a visible and palpable bony protuberance in the right parietal region measuring about 4 cm. in its transverse diameter. The left arm and hand were slightly smaller than the right but functioned normally. From

the tip of the acromion to the tip of the middle finger the left arm measured 2 cm. less than the right. The left arm was definitely weaker than the right. The left elbow and knee reflexes were slightly stronger than the right. Other reflexes were normal. Sensation was normal except for the anesthesia over the lateral half of the volar surface of the right hand exclusive of the thumb, evidently the result of a recent injury. Ophthalmologic examination gave negative results except for an alternating squint. Roentgenograms showed a definite thickening of the skull in the right parietal region with what appeared to be the remains of an old stellate fracture about 4 cm. from the midline near the fissure of Rolando.

A diagnosis of jacksonian epilepsy, probably the result of a depressed fracture of the skull, was made. Since the patient was right-handed and there seemed to be little danger of producing new symptoms by an operation, an exploratory craniotomy over the right rolandic area was done on June 28, 1928. An osteoplastic flap was turned down. Some difficulty was encountered owing to the depression and thickening of the bone in the region of the injury. The dura was adherent at this point, and there were a number of irregular spurs of bone on the dural side of the flap. Although no undue bleeding had been encountered, the patient showed signs of shock and the operation was discontinued. On July 5, the patient having made an excellent operative recovery, the stitches were removed and the flap separated by blunt dissection. A soft area was found in the anterosuperior portion of the wound beneath a thickened, scarred dura. With an aspirating needle, a brownish turbid fluid was withdrawn. The opening was widened, and a cyst about the size of an English walnut, with a thin smooth lining, was found. An opening was accidently made through this exceedingly thin wall, exposing the lateral ventricle and furnishing an excellent view of the foramen of Monro. A considerable amount of clear spinal fluid escaped. The dura posterior to the cystic area was firmly adherent to the brain. It was freed and removed for a distance of about 3 cm. The brain beneath this was whiter than the surrounding tissue. The irregularities on the inner surface of the bone flap were carefully removed, and the flap replaced without an attempt to close the dural defect.

The following day, the patient began to have a septic fever, the temperature varying from 101 to 103 F. The wound in the scalp remained clean, but the dressings were saturated with cerebrospinal fluid and required daily changing for about eight days. On July 16, eleven days after the operation, the patient had a positive Kernig sign. He began to vomit, and the optic disks appeared slightly blurred. An unsuccessful attempt was made to reestablish drainage from the wound. The patient's condition remained practically the same, except for a slight stiffness in the neck, until July 19 when, at Dr. Bassoe's suggestion, a spinal puncture was made and 30 cc. of turbid fluid was withdrawn. There was no marked increase in pressure. This fluid showed: 4,780 cells per cubic millimeter, with both polymorphonuclear leukocytes and lymphocytes present; a positive Nonne test, and a gold chloride curve of 0013321000. In smears, a gram-negative, short bacillus was found. Cultures showed the same organism, which culturally had all the characteristics of *B. fecalis alcaligenes*. The patient's temperature dropped immediately after the spinal puncture and stayed normal for the ten days he remained in the hospital. He had had a number of attacks preceding the spinal puncture and none afterward. The patient went for more than six months without any recurrence of symptoms.

I have been unable to find in the literature any record of a case of meningitis due to *B. fecalis alcaligenes* and little about other infections produced by this organism. According to Stitt (Practical Bacteriology, Blood Work, Parasitology, ed. 8, Philadelphia, P. Blakiston's Son & Company, 1927, p. 179), the organism is a frequent inhabitant of the intestinal tract. It has been isolated in the blood in a few cases resembling typhoid fever. Fürth (*München. med. Wchnschr.* **9**:2669, 1913) reported six cases of typhoid-like disease in which *B. fecalis alcaligenes* was obtained and in which agglutination occurred for this organism but not for those of typhoid or paratyphoid. The organism is usually considered nonpathogenic, and Kendall (Bacteriology, ed. 2, Philadelphia, Lea & Febiger, 1921,

p. 315) stated that animal experimentation has been uniformly negative. As it culturally and clinically resembles the organism of paratyphoid, I looked over the literature on paratyphoid meningitis and found that Bradhy (*Arch. Pediat.* **42**:550 [Aug.] 1925) reported the first case in the English literature and cited seven others, one by Walterhöfer (*Deutsche med. Wchnschr.* **43**:1036, 1917) being of particular interest. This case occurred in a soldier, aged 20, who received a minor cranial injury. *Bacillus paratyphosus* B. was obtained in pure culture from fluid by lumbar puncture, with more than 3,000 leukocytes and lymphocytes in a turbid spinal fluid. Following the third spinal puncture, the patient's temperature dropped immediately to normal and he went on to a good recovery.

DISCUSSION

DR. ALBERT B. YUDELSON: Was a blood culture made in this case?

DR. L. C. GATEWOOD: A blood culture was negative at the time of the operation. It is still possible to get the patient's serum and test it for agglutination with *Bacillus fecalis alcaligenes*. In all reported cases of blood-borne infections, the agglutination is not high, usually not higher than 1:50.

DR. LOYAL DAVIS: This case is interesting, because one has been taught that the organism is nonpathogenic.

EXPERIMENT ON CLAUDE BERNARD'S PIQÛRE. DR. FRIEDRICH HILLER and DR. A. TENNENBAUM.

This article will appear in full in a later issue of the ARCHIVES.

EXPERIMENTAL STUDIES CONCERNING THE NERVOUS REGULATION OF SUGAR METABOLISM. DR. FREDRICH HILLER and DR. ROY R. GRINKER.

This article will appear in full in a later issue of the ARCHIVES.

NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY AND OF OTOLOGY

Joint Meeting, Jan. 11, 1929

JUNIUS W. STEPHENSON, M.D., *Chairman*

TREATMENT OF OTOLOGIC ABSCESS OF THE BRAIN. DR. HARRY P. CAHILL (by invitation).

This paper will be published elsewhere.

NEUROSURGICAL TREATMENT OF ABSCESS OF THE BRAIN. DR. JOSEPH E. J. KING.

Abscess of the brain occurs most frequently, according to location, in the following order: temporosphenoidal, cerebellar and frontal. The most favorable case for operation, and one in which there are no unfavorable sequelae, is the abscess of the temporosphenoidal lobe, especially if it is encapsulated. The next most favorable is the abscess of the frontal lobe, and the least is the cerebellar abscess.

In dealing with intracranial complications, especially those associated with infections of the sinuses and the mastoids, one should be careful to differentiate between an epidural abscess and an abscess of the brain. This differentiation is not always easy, especially in the early stages. Intracranial explorations have been performed with negative results because the lesion is extradural. When in

doubt it is advised that an operation should be done on the probable source of infection, for example, the mastoid, the frontal sinus, etc.

Although various forms of drainage materials have been advised and used in operations for abscess of the brain, none of them adequately drains in the true sense. The most logical drain is the conical wire mesh drain (Mosher). It is believed that the residual portion of the abscess cavity "plasters" itself against the drain and then herniates through the opening in which the drain was inserted, thus effecting a cure. However, not all these abscess cavities will collapse symmetrically about this cone-shaped drain; in such cases secondary pockets, or abscesses, will surely develop. I believe that it is advisable to discard drains, as such, entirely, and instead allow the abscess cavity to herniate completely from the intracranial space through a bony opening, made sufficiently large and so placed as to allow complete herniation. By so doing there is nothing to be drained, because the abscess cavity has completely everted itself and has become the dome of the hernia cerebri; infection to the meninges is unlikely, the increased intracranial pressure is immediately lessened, and there is no danger of the formation of a secondary abscess or rupture of the abscess into the ventricle. One or more of the conditions enumerated, if not prevented, are fatal. It is, therefore, advised that: 1. The exact localization of the abscess should be made by the use of a brain canula through a trephine opening. 2. The trephine opening should then be enlarged so as to correspond in position, size and location with the abscess cavity, i. e., the opening should be so enlarged that it is directly over the abscess. 3. Complete herniation of the abscess cavity should be secured so that no drains are required. 4. Dakinization of the area should be maintained until the hernia cerebri has subsided. 5. Epithelization of the area is needed.

In dealing with an abscess of the brain, one should realize that there is only the faintest possibility of saving life and should disregard statistics completely, i. e., one must take the bad with the good and operate on any type of abscess in which there is the slightest chance of recovery. Cases in which there is a concomitant, severe meningitis are poor surgical risks. It is believed that a higher percentage of recoveries would follow an operation that is delayed than one which is done immediately during the severe meningitis. All cases of encapsulated abscess of the brain have evidently passed through a period of more or less severe meningitis, and the patients were able to survive until encapsulation could take place.

Six patients whom I have operated on by this method have recovered, the first operation having been done in 1920. The patients in several other unfavorable cases have died. Two patients died because an operation on the frontal sinus was not performed sooner. Two additional cases may be reported: one an abscess of the frontal lobe, and the other an abscess of the temporosphenoidal lobe. A plea is made for more hearty and thorough cooperation between the neurolgist, otologist and the neurosurgeon.

DIAGNOSIS OF ABSCESS OF THE BRAIN. DR. FOSTER KENNEDY.

In the absence of etiologic factors pointing to a source from which abscess formation could take origin, the diagnosis of abscess of the brain is almost impossible. One can from certain signs and symptoms postulate the presence of an expanding lesion and its location; but in the absence of ear disease, sinus disease, trauma, or pus elsewhere in the organism it would be rash to make a diagnosis of abscess on the mere speed of expansion, because almost the quickest and the most acute expanding lesions within the brain are gliomas into which a hemorrhage has occurred; and they often simulate an abscess. Fever is almost always absent in cases of abscess unless the meninges are involved; in my experience, disproportionate drowsiness is the most general diagnostic feature of an abscess. The hebetude and stupor accompanying abscesses of the brain are usually out of all proportion to the degree of intracranial pressure, whereas in tumor the amount of stupor is usually rather less than one would expect from the tremendous amount of edema, headache, vomiting and other symptoms. Some of you saw a girl,

aged 15, in the Bellevue Hospital with a papilledema of about 7 diopters. I saw her at Christmas; she was then comfortable and apparently happy, which would have been impossible in any case of abscess of the brain. A generalized headache with local emphasis is important in the course of chronic or acute otitis media, but increasing drowsiness is a still more important feature. Papilledema is uncommon in abscess of the brain, though a slight change in the fundus is usual; a little obscuration of the disk may be seen at the nasal margin, but frank papilledema is almost unknown, probably because the disease is too short and ingravescent to permit it to develop. An abscess of the brain can exist in a static condition for years if encapsulation occurs. In that event, no papilledema occurs; papilledema is not an inflammation but a mechanical situation whereby the cerebrospinal fluid is forced down the vaginal sheaths of the optic nerve, etc.; if enough pressure occurs, the papilledema does not appear in abscess of the brain. An abscess may expand rapidly or may be operated on before papilledema occurs; but that does not occur in tumor, where the course of the disease is much longer.

The diagnosis of abscess is fairly difficult in the absence of a direct lead from the etiology. I will cite the case of a patient who was under my care many years ago in the Bellevue Hospital. He was admitted with a diagnosis of paresis; he had much mental change and loss of memory, and was syphilitic. He was thought to be paretic probably because one pupil behaved unusually. I found slight pallor in the outer border of one disk, and papilledema developed in the other eye. He rapidly became blind in the eye in which there was a little pallor but no papilledema, and although there was no history except of being wounded "in the scalp" two years previously, I made a diagnosis of abscess in the left frontal lobe, on the side on which he had lost his vision. Dr. Rogers operated—in the face of a good deal of opposition—and removed 10 ounces of pus from the right frontal lobe. The reason for the loss of vision was that the abscess was so large and so old that it had come to press on the right optic nerve, as a tumor might have done; this prevented fluid from being forced along the vaginal sheath and damaged the macular bundle of the optic nerve. That situation not having occurred in the opposite eye, there was papilledema. That diagnosis of abscess of the frontal lobe was made in terms of an expanding lesion of the frontal lobe. I do not recall that I made a diagnosis of abscess or tumor, but rather of a frontal lobe situation. The improvement that occurred in the man's mental condition was remarkable, and no alteration in his personality could be noted a year after the operation, in spite of the fact that the right frontal lobe must have been seriously affected by so much pus.

In a series of seventy-six abscesses of the brain, fifty-five were found in the temporal lobe, thirteen in the cerebellum, four in the cerebrum, two in the pons and one in the cerebral peduncle; therefore, the symptomatology of the temporal lobe is of prime importance to the otologist, apart from the general diagnosis of abscess, the symptoms of which are not much more than increasing headache and drowsiness. The temporal lobe may contain a gigantic abscess without many localizing signs. However, the lobes occasionally are irritated by abscesses and then give rise to bursts of subjective sensations of taste and smell. I say bursts as these are in the nature of release phenomena like jacksonian epilepsy—a "burst" of bad smell or a "burst" of bad taste, etc. Then, there are curious psychic phenomena which occasionally come with an abscess though they are on the whole more likely to be described in cases of tumor. The hebetude of the patients prevents an expression of these psychic features—dreamy states, sometimes hallucinations of figures, animals, birds, flowers and people which are recognized by the patient as hallucinations. I saw a patient in the hospital with a tumor of the left temporal lobe, who every now and then saw in his room a man of whom he said, "I believe I had had a business transaction with him"; i. e., the figure, although unidentifiable and not capable of being described by him, yet had a curious intellectual attachment for him, for the figure was familiar. He could not say whether the man wore a hat, whether he had a beard or was clean shaven—but he saw the figure and felt as though he "knew he had had a business transaction with him." That shows the extraordinary intellectual projection that occurs with these voluminous mental states.

In another case, the patient, a woman, said that she saw quite suddenly men and women dressed in eighteenth century costumes; the women were in full skirts, with low bodices, and the men in knee breeches and ruffles; they were crossing from left to right and talking to one another. "Did they look real?" "Yes." "Did you think they were real?" "No." "How did you know?" "I knew they were not real, for they were out of place in a New York apartment!"

Such extraordinarily acute visions always occur in the presence of organic disease in the temporal lobe. One may wonder why such odd memory projections should take origin from diseases of the temporal lobe. After all, we are each of us microcosms of the race, passing through all its stages in our evolution from egg to adult. Lower forms have much of their intellectual attachments through smell centers, i. e., the temporal lobes. It may well be that in fetal and infantile life one uses these areas to store memories—a function late in life taken over by a more highly organized, more recently acquired mechanism, placed perhaps frontally.

The aphasia in cases of abscess, which occurs mostly when the disease is on the left side of the brain, is nearly always wrongly described. These patients are not word-deaf; they understand what is said to them; they are word-forgetful. They cannot recall exact memories of words. If one tries to make them say the wrong word, they resent it—therefore they are not word-deaf, but they are word-forgetful and call things by wrong names to their own chagrin. They cannot find the right word, though they often find a word something like the one they want; they often get a word beginning with the same letter, or one which carries a cognate meaning. It is really an anomia and sometimes, if not too stuporous, patients will talk brilliantly in a jargon. Hemianopia occurs if Meyer's loop is impinged on. In tumors interference with the visual field will take place, not in form but in color, before there is deterioration of appreciation of form. That is something that has to be looked for.

Patients with cerebellar abscesses, as Dr. King has said, nearly always die. The lobulation of the cerebellum curiously facilitates the running of the pus in different directions, so that an abscess once situated in that lobe usually has diverticula. One can detect the location of the lesion by the ataxia, not of station but of limb; ataxia of station, vertigo and nystagmus, and vomiting may all be labyrinthine, they may not be cerebellar at all; add hebetude to the picture and ataxia not of station but of limb, and, especially with papilledema, one will then have a picture of cerebellar abscess.

In cases of epidural abscess there is almost no rise of intracranial pressure, and hemianopia is practically never present. The local signs are often near the ears—the attitude and fixation of the head is important. Sinus thrombosis can usually be diagnosed by the high fever, the chills, the local jugular complications and the changes in the blood picture.

Probably the most difficult differentiation to make from an abscess of the brain is diffuse meningitis, which is sometimes impossible to distinguish from a localized abscess. In one case I was certain that the patient had an abscess in the frontal lobe, and yet he had a normal spinal fluid. There was a rigid neck and Kernig sign with papilledema, hebetude, etc.; in spite of the meningeal signs the spinal fluid was normal. An operation was done and an abscess found, but there was also a meningitis which had so glued the spinal membranes near the atlas that the spinal space was completely cut off from that in the head; examination of the lumbar fluid was therefore of no value.

I wish to congratulate Dr. Cahill and Dr. King on their success in this very difficult and courage-requiring field of surgery. Many surgeons shy away from abscess of the brain, largely because of the mortality rate, but I have seen much of Dr. King's work and know that he will attack any case of abscess confronting him, or any meningitis that might conceal an abscess.

DISCUSSION

DR. EDWARD B. DENCH: During the last seven years, at the New York Eye and Ear Infirmary we saw 94,469 ear cases of all grades of severity, and among

these we found only eleven cases of abscess of the brain. Personally, I am inclined to believe that the proportion should be much larger; in the hospital reports the cases of abscess include only those recorded as operations for abscess of the brain. These figures may be contrasted with another series of cases which I analyzed several years ago. There were 10,000 or 15,000 cases of acute and chronic suppuration of the middle ear, and eighty-five of these patients had some intracranial complication: abscess of the brain, sinus thrombosis, meningitis or extradural abscess. While a fairly large series of cases of suppurative lesions of the middle ear will show over 1 per cent of intracranial complications, the incidence of abscess of the brain in a large number of ear cases examined is extremely low.

My experience with abscess of the brain consists of twenty-seven patients operated on either by myself or by a member of my staff. Of these, twenty-one were cerebral and six cerebellar abscesses. Nine of the patients with cerebral abscesses were cured, and twelve died. One of my first patients was hale and hearty a few months ago despite the fact that he had had a large abscess in the temporosphenoidal lobe thirty years before. There were six cerebellar cases; four patients died, and two were cured. Contrast these statistics with those reported by Macewen in his work on "Pyogenic Diseases of the Brain and Spinal Cord," in 1893. He reported nineteen cerebral abscesses with eighteen recoveries and one death. It should be remembered, however, that all of Macewen's cases were not of otitic origin. Naturally, traumatic cases present an entirely different aspect from those of otitic origin. All of my cases were of otitic origin. Korner reported fifty-five cases of cerebral abscess with twenty-nine recoveries and twenty-six deaths. This represents the average mortality in otitic abscesses of the brain.

In 1907, I collected and reported in the Transactions of the American Otological Society 100 cases of cerebral abscess; fifty-two of these patients were cured, and forty-eight died.

Regarding the cerebellar abscess, Macewen reported four cases in which all the patients recovered. These were undoubtedly traumatic cases. Korner reported fifty-five cases with twenty-nine recoveries. In the Transactions of the American Otological Society, 1907, I gave a collated report of 102 cases of cerebellar abscess with sixty-nine deaths and thirty-three recoveries. This is a rather high mortality, and I was much comforted to hear Dr. King say that most of the patients with cerebellar abscesses died.

I think that the Mosher wire drain is an excellent device, but, unfortunately, I have had few cases of abscess of the brain since this was put at the disposal of the profession; these have all resulted fatally, not on account of the Mosher drain but in spite of it. I think that the actual drain used is of little importance. Experience has taught me that one thing to be avoided is too frequent dressing in these cases. I remember that it was the custom in my early cases to put the gloved finger into the brain at each dressing so as to search out and evacuate any secondary collection of pus. To my mind the gloved finger, if properly educated, will do much less damage to the brain substance than any mechanical device. Macewen's work teaches that the less the brain is manipulated the better the result. If you read his reports carefully, you will find that he opened the abscess, inserted the drain and did little after that. If the patient does well after the abscess has once been drained the dressing should be changed infrequently. I prefer an ordinary cigaret drain carried far into the abscess cavity; as the brain tissue expands it pushes this drain out, and one can replace it later by a thinner drain of gutta-percha tissue. Many other methods of drainage are probably just as good.

Whether one should operate through the avenue of infection or through a sterile area is worthy of discussion. Of 100 cases of temporosphenoidal abscess to which I have referred, thirty-seven patients were operated on through a sterile area; eighteen were cured, and nineteen died. Forty-one were operated on through the avenue of infection; twenty-seven were cured, and fourteen died. Operating through the avenue of infection, therefore, seems to afford better results. When one is certain that the abscess is on the left side, statistics seem to show that the prognosis is a little better when the operation is performed through the infected

rather than through a sterile area. When the abscess is on the right side of the brain and without localizing symptoms the operation should be done through a sterile area above the zygoma. In a left temporosphenoidal abscess, by operating through the infected area one is able, almost always, to find the stalk of the abscess; this represents the avenue of infection, and has caused a walling off of the arachnoid space over a considerable area. By operating through the stalk of the abscess there is little danger of meningeal infection; on the other hand, if the abscess is drained through an area where obliteration of the arachnoid space has not occurred, a fatal meningitis is most likely to be the result. With proper technic, inspection through a sterile area is a safe procedure. My colleagues felt certain that a temporosphenoidal abscess was present in one case at St. Luke's Hospital, but I did not agree with the opinion. The patient had had a serious suppuration of the middle ear with sinus thrombosis, and the mastoid wound had not entirely healed. In this case I isolated the suppurative area and exposed the temporosphenoidal lobe above the zygoma. Exploratory puncture was done with negative results; the wound was closed and the man suffered no ill effects from the operation, and lived for several weeks after the exploratory procedure. This shows that the brain can be explored through a sterile field without fatal results.

Regarding the cerebellar abscesses in this series, forty-five were opened behind the lateral sinus; twenty-five cures and twenty deaths resulted. Operations were done in front of the sinus in eleven cases; four ended in recovery, and seven in death.

As to the two-stage operation for cerebellar abscess, I may mention the case of a patient who had had a simple mastoid operation which did not heal; he developed vertigo. He had a dead labyrinth on the affected side; the spinal fluid was under pressure and showed 15,000 cells per cubic millimeter. I operated the same evening, doing a rapid radical operation and extirpating the labyrinth and removing the posterior surface of the pyramid down to the internal auditory meatus; I removed the floor of the middle cranial fossa, thus exposing Trautman's triangle thoroughly. The patient had a slight but definite nystagmus in both directions before the operation. Although I felt convinced that a cerebellar abscess was present, I hesitated to open the dura because the field of operation was so foul. As nystagmus persisted to the affected side in spite of the extirpation of the labyrinth, two days later I entered the cerebellar substance through the sacculus endolymphaticus and drained the cerebellar abscess. The patient is now well, nearly two years after the operation. I think that entering the brain substance at the primary operation in that case would probably have caused a fatal result. I also wish to point out the great importance of nystagmus to the affected side on the diagnosis. The persistence of nystagmus to the affected side made the diagnosis certain in this case.

Dr. Kennedy stated that fever does not occur in cases of abscess of the brain. This is true of the stage in which the neurologist sees these patients and in fact of the stage in which most of us see them. There is, however, in most cases a distinct stage of invasion, and during this stage the temperature will range from 102 to 104 F., with a high white cell and polymorphonuclear count. This stage frequently escapes observation as it is of short duration, but it should be remembered that a rise in temperature after a simple mastoid operation may mean one of two things: an ordinary postoperative rise, or an acute invasion of the cerebral tissues; this possibility should always be remembered. I disagree with Dr. Kennedy that one should wait for the subsidence of a papilledema before operating for abscess of the brain. The presence of papilledema indicates increased intracranial pressure and is an important indication for immediate operation. To wait until the swelling of the optic disk has diminished may sacrifice the lives of many patients.

Dr. Ira Cohen: In this diagram I have drawn a circle representing an abscess cavity and in it a smaller circle representing a drainage tube. If one is fortunate, the larger circle will contract down uniformly on the smaller as would a rubber ring. However, the contraction may not be uniform. My second diagram shows

the outer circle irregular in appearance like a crenated red blood corpuscle. In my third illustration the irregularity in contour is more marked, and the opposite sides of the abscess cavity touch. The next diagram reveals the opposite sides agglutinated, leaving one cavity drained by the tube and the second cavity completely sealed off and not drained. This was brought out clearly by iodized poppy seed oil 40 per cent injected into the abscess of the frontal lobe secondary to an ethmoidal infection. X-ray pictures taken several days after the injection showed that the iodized oil had not drained out, and a secondary abscess had formed which had to be drained. I believe that this happens frequently. I have not used the metal drains which Dr. Cahill mentioned, but if the abscess cavity contracts down about them they would tend to prevent such secondary abscesses.

Cerebellar abscess takes a sad toll. Within the past few months I have had under observation a patient who I thought was going to recover. The boy was discharged from the hospital apparently well except for a residual papilledema. He returned in three weeks; the abscess was drained again. A third operation for drainage had to be done, and the boy died. I think that the occurrence of a secondary abscess by pinching off from the primary one is often a reason for poor results.

Dr. William Alfred Scruton: The apparent advantages of the form of drain shown by Dr. Cahill are several, the outstanding being that it remains in position and that the precentage of recoveries is unusually high.

In this connection I would direct attention to the difficulties encountered in cases of multiple abscesses. Two such patients have recently come to my attention, one of my own in whom three distinct abscesses were located at the same operation and another of the late Dr. Shepard, of Brooklyn, in whom five abscesses were located. The first patient was discharged as cured; however, the symptoms returned four months later when another abscess was located and drained. The patient did not recover. At the original operation the multiple abscesses were located by digital examination. The accomplishment of exploration of the brain for abscess may be facilitated by the use of a modified form of the ordinary bayonet forceps as developed by Dr. John R. Page. This instrument is available for inspection at the Manhattan Eye, Ear and Throat Hospital.

Dr. J. L. Maybaum: If one may judge from the diversity of the procedures proposed for the treatment of abscess of the brain, the last word on this problem has not been said. Two years ago, a prominent neurosurgeon read a paper before the Otological Section on the treatment of abscess of the brain. In brief, his procedure was to make a small trephine opening over the location of the abscess, aspirate the contents and do nothing more unless further symptoms presented themselves in which event this procedure was repeated. He cited cases of recovery from this method. One may assume that in each instance the abscess was small and well localized, and that both the patient and the surgeon were fortunate.

From this simple procedure to that followed by Dr. King are extremes in the surgical methods. I believe that until Dr. King proposed his operation, the various operations done were much alike. While I hardly believe that Dr. King's operation is indicated in all cases of abscess of the brain, I am inclined to regard his method as a decided step forward. I have had experience with various materials used for drainage, rubber tubing, dam or gauze; they are frequently ineffectual. Such drainage frequently does not accomplish the purpose intended; effective drainage does not take place; secondary abscesses not infrequently occur. There is little knowledge of what is happening within the wall of the abscess. By means of Dr. King's operation the abscess wall is before one's view at all times. Would Dr. King advise his operation in a deepseated abscess, which, because of its location, may not herniate? Would he perform his operation in a left-sided temporosphenoidal abscess or in a cerebellar abscess?

The neurologist can be of help in determining the time of operation. An acute abscess, before an attempt at walling off has occurred, may result fatally if operated on too early. The time has come when the neurosurgeon, neurologist and otologist must cooperate actively in order to reduce the high mortality in cases of abscess of the brain.

DR. CAHILL: Dr. King is right about the dangers of secondary infection in abscesses of the brain. The abscess does not force the capsule out, and secondary symptoms or exacerbation of the old abscess or reinfection is possible. I have operated on those. And yet, one of the pictures of the iodized oil that I demonstrated showed that the iodized oil was still present in the brain one year after the operation. That brain capsule did not follow the wire drain out as we wished, or else the iodized oil entered the brain through the abscess cavity.

Dr. Dench spoke of after-treatment. I think that one does dress abscesses too frequently. I think a rubber dam with petrolatum on it, or petrolatum-protected as much as possible, is of great importance. Recently, Cushing dissected out the frontal lobe in toto with perfect recovery. I do not know whether any other cases have been reported with recovery or not.

In 1925, I reported three cases of cerebral abscess with recovery. Since then I have had one. I think it is important to drain through the avenue of infection. If you do not clean it, and if the infection is through the labyrinth and you do not drain through that area you are not going to work through a protected meningitis. If it is through the sinus and you do not work through it you are not going to work through a protected area. The abscesses are not as chronic as those of the cerebrum, and hence the protective material may be rather virulent.

I was thankful to hear Dr. Cohen. I needed him three months ago when I was speaking in Boston as regards the development of an abscess, and the capsule collapsing and walling off other abscesses.

As regards the number of cases in the Eye and Ear Infirmary in Boston during the last two years, we have had twelve cases of abscess of the brain, three of which were discovered at autopsy. I think that until we have more necropsies we will not be sure of abscesses. In so many of the cases, operation had not been performed.

As regards operating through the sinus, I think that that is a point that often is not given ample consideration. Walling off the sinus and operating through the sinus will often give a much better area to work through than Trautmann's triangle. I think that that is a point to which insufficient consideration has been given in otologic work. Cooperation with the neurologic surgeon should be always present. Any man who attacks a brain abscess should at least be an embryonic neurosurgeon.

DR. C. D. KING: One thing Dr. Cohen brought out about the development of secondary abscesses, I have seen several necropsies in which just what he spoke about had taken place, and that is one reason I gave up using the drain.

I certainly enjoyed Dr. Dench's review of the subject. Dr. Maybaum's remarks about not operating too soon were pertinent. A patient with an abscess so encapsulated that it can be plucked out as Dr. Scruton described, naturally has gone through a serious time before being seen by the otologist, probably fortunately for the patient, because most likely the otologist would have operated on him. The few that do encapsulate have fortunately had enough resistance, or enough something to allow them to develop to a stage when one can do something more readily for them.

Dr. Maybaum also spoke of the help of the otologist, or the rhinologist, or whoever operates on the sinuses and mastoid. The two patients I had certainly should be living today if there had been a man who knew what to do with these sinuses at the proper time. Both of them died from abscesses formed by direct extension.

In a case of localized abscess without violent fever and all other meningeal signs and symptoms, if it is localized, whether encapsulated or not, if properly attended to from the otologic, laryngologic or rhinologic point, i. e., if the focus from which the infection originally took place, is well cared for, I believe that the surgical results are better than in practically any case of a tumor of the brain other than a meningioma.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 25, 1929

N. W. WINKELMAN, M.D., *President, in the Chair*

PROGRESSIVE PALLIDAL DEGENERATION: A NEW CLINICOPATHOLOGIC SYNDROME. DR. N. W. WINKELMAN.

This paper, the presidential address, will be published in full.

Dr. Winkelman presented a new clinical syndrome appearing in two brothers, the outstanding manifestations of which were marked rigidity without hyperkinesis, and an associated retinitis pigmentosa. An autopsy was obtained in one case, and neuropathologic studies disclosed a peculiar degeneration limited to the pallidal system. A search of the literature failed to disclose a similar case of degeneration so definitely confined to the pallidal system. Certain of the clinical symptoms were thought to have been produced by the degeneration noted and may thus help to clarify the physiology of this most complicated portion of the nervous system. Slides illustrating the degenerative changes were shown.

DISCUSSION

DR. WILLIAM G. SPILLER: Dr. Winkelman has described the purest known case of degeneration confined to the pallidal system; other similar cases in the literature are more complex symptomatically and the pathologic observations implicate other structures as well as the pallidum; this is notably true of the cases reported by Hallervorden and Spatz, and Kalinowsky. In Dr. Winkelman's case there were no mental symptoms and no pathologic changes in the cortex.

Dr. Winkelman states that no symptoms of disease of the pyramidal tract were present in his case. It might be interesting to have his explanation of the exaggeration of tendon reflexes. Tremor was not present, and increased muscular tonicity in the form of rigidity was the pronounced symptom. Spasticity has been distinguished from rigidity, partly by differences in innervation of agonistic and antagonistic muscle groups and by differences in reaction after stimulation of muscles. It would be interesting to have Dr. Winkelman's view of increased tone as caused by disease of the pallidal system, which in his opinion consists of the pallidum and the zona reticulata of the substantia nigra. He states that the pallidum has to do only with tone. Does he intend to imply that all increase of muscle tone depends on disease of the pallidal system, and does disease of this system act directly in producing increase of muscle tone or by loss of the regulating influence of this system? He refers, however, to a case of epidemic encephalitis with rigidity, and in this the zona reticulata was intact.

He has described the greatest degeneration in the innermost portion of the pallidum, and he seems to suggest that the greater involvement of the lower limbs may be related to the observations in this inner segment; his views, however, are expressed with considerable reservation. He speaks of the degeneration in his case as an abiotrophy, and compares it with progressive spinal muscular atrophy. The comparison seems acceptable. He refers to the rigidity without tremor occurring in carbon monoxide poisoning with degeneration of the pallidum. This was the condition in the case of carbon monoxide poisoning reported by McConnell and me in 1912. Microscopic examination showed pronounced degeneration of the pallidum.

Reference has been made to the relation of Huntington's chorea to degeneration of the striatum and cortex, and especially to Kinnier Wilson's views regarding the striatal origin of this disease. It is important in this connection to refer to Wilson's recent presentation at the meeting of the Society of German Neurologists. He emphasized that there are two groups of symptoms: those which depend on a loss of function and those which depend on the loss of inhibition of function. It is to the latter group that he devotes his attention, and in this he includes chorea,

athetosis and other forms of hyperkinesis. He does not accept the view held by many, viz., that chorea may be explained by lesions of the striatum. He emphasizes a statement of Hughlings Jackson that destructive lesions can never produce increased function directly. The question is not what mechanism is paralyzed, but what mechanism is functioning. Wilson believes that chorea is manifested through the corticospinal (pyramidal) system, and this system must be relatively intact or hyperkinetic symptoms cannot appear. This statement seems almost self-evident.

Wilson studied a case of senile chorea during a period of eleven years, and the patient died at the age of 88. Severe choreiform movements affected only the right side. The cerebral cortex was in good condition except the left postcentral convolution, and this was atrophied to one half the normal size. The left corpus striatum was intact by microscopic examination. This case, Wilson believes, refutes the striatum theory of the causation of chorea. He attributes great importance to the cortical atrophy in his case. Chorea or athetosis develops when the afferent tract from the cerebellum through the subthalamic region and optic thalamus to the cerebral cortex is impaired. Wilson assumes that because of the atrophy in his case certain changes were produced in the sensorimotor cortex which disturbed the regulation of the motor stimulation by the transcortical and cerebellocerebral tracts.

Marburg, in discussing this paper, expressed the opinion that any lesion which implicates the cerebellocerebral tract may produce these involuntary movements without cortical involvement. Wimmer believed that the corpus striatum could not be dismissed as related to involuntary movements.

Any one unfamiliar with Wilson's previous work would get the impression from this address that Wilson denies all relation of the striatum to hyperkinesis, but he has in earlier papers stated that he believed that the corpus striatum exerts a regulatory influence on movement.

A few years ago we had another remarkable presidential address on a similar topic by Dr. Charles M. Byrnes. In this painstaking investigation the degenerative changes in the muscle spindles were found in parkinsonism. The relation of these observations to the rigidity from disease of the pallidal system must be determined, and from the combined picture we may have a proper conception of the disorder.

Dr. F. X. Dercum: Retinitis pigmentosa has been found associated with many abnormal states, such as general retarded physical development, failure of full development of the extremities, polydactyly, talipes varus, harelip, microcephaly, idiocy, epilepsy, mental subnormality, deaf-mutism, speech disturbances and with dystrophia adiposogenitalis. In addition, various abnormalities of the eyes have been noted, such as micro-ophthalmus, keratoconus, persistent hyaloid artery, dislocation of the lens, high grade myopia, strabismus and nystagmus.

To these observations are now to be added the pallidal degeneration revealed by Dr. Winkelman's careful studies. Evidently, one has to deal here with a disease featured by a marked biologic subnormality and deficiency; imperfect developments not only of the nervous system but apparently of other structures as well. Everything about the disease suggests a biologic level definitely and decidedly below the normal. This fact cannot, I think, be overemphasized.

In addition to the symptoms detailed by Dr. Winkelman, the patient, whose case he studied at autopsy, presented, as among the suggestive features of his case, a distinct and unmistakable enlargement of the thyroid gland. Enlargement of the thyroid is time and again associated with thymic persistence. Such thymic persistence may be only slightly present and not sufficient to betray itself clinically. Indeed, unless the thymus gland is enormously enlarged, the roentgen rays give no help in determining its condition. It is only when it projects in the intercostal interspaces at the edge of the sternum that it can be observed. The lessons that are taught by autopsies in exophthalmic goiter, in which, in my experience, the enlarged thyroid is always accompanied by a persistent thymus, justify the inference that the enlargement of the thyroid is merely an effort of nature to compensate for a persistent thymus, a thymus that has never undergone involution.

A further observation in the case which has not been mentioned by Dr. Winkelman is that the blood revealed a lymphocyte count of 32 per cent. The patient was already 24 years of age and his lymphocyte count should have fallen to 18 or 20 per cent, that of the normal adult period. Such an excess of lymphocytes is suggestive of thymic persistence and would account for the somewhat enlarged thyroid. The study of the blood suggests an infantile condition and is itself an evidence of retarded development, just as much as are any of the other factors presented by the case.

A question that presents itself is: Did the pallidum undergo degeneration, did its cells undergo destruction and death, because of biologic failure or inadequacy, or because of an imbalance of the glands of internal secretion or some other autotoxic factor concerning which we have as yet no evidence? Possibly all of these factors played a rôle.

As regards the symptom of spasticity and rigidity, the following appears to me to be the explanation: If the pallidum is destroyed, one certainly cannot attribute the increased tonus of the muscles to the pallidum, but the cause of the increased tonus must be sought in the motor cortical innervation. In fact, this is the only possible explanation which biology — comparative anatomy and embryology — justifies. In the fish, the pallidum is the only structure from which a motor pathway arises. In the higher vertebrates, a cortical motor innervation is added. These two innervations of the muscles, one from the motor structures of the paleoencephalon and the other from the motor centers of the cerebrum, mutually inhibit each other. If the pallidum is destroyed, there is a rise in tonicity due to the now unopposed action of the corticospinal (pyramidal) pathway; on the other hand, if the motor cortex or pyramidal pathway is destroyed, there is a rise in innervation due to the now unopposed pallidal innervation. In the first instance, one has the spasticity of the parkinsonian symptom-group; in the second instance, one has the spasticity seen in an ordinary hemiplegia.

The pallidum can be regarded only as a motor organ, as an organ in effector relation with the rubrum and the motor neurons in the spinal cord. It may, of course, be in affector or receptor relation to the pallidum and caudatum, i. e., with the neostriatum, but as regards the rubrum and the cells of the anterior cornua of the spinal cord it is an effector organ. The biologic facts in our possession permit of no other explanation.

DR. N. W. WINKELMAN: Dr. Spiller has given me some difficult questions to answer. The exaggeration of the tendon reflexes was probably dependent on the increase in the muscle tone. There was no doubt that in this case the pyramidal tract was intact. It might be of interest to recall that not only may there be an increase in the reflex activity in extrapyramidal disease, but even clonus has been described. There was no intention of limiting the center controlling tone to the pallidal system for I am well aware of the fact that with involvement of the substantia nigra marked increase in tonus occurs. I did refer to a case in which there was such marked increase in tonus that the patient could be moved like a piece of wood, and microscopic examination in this case has shown that there was complete destruction of the compact portion of the substantia nigra bilaterally. Other centers may also be responsible.

Dr. Spiller has well sensed my great degree of reservation in even suggesting that the three anatomic segments of the pallidum might have something to do with the innervation of segments of the body. I made this suggestion about a year ago and recently have come across an article by Schilder and Stengel in which the same suggestion is made. The one thing that I do want to stress is that with such marked differences in the origin and development of the pallidum, as contrasted with the striatum, it does not seem logical to me to carry over the same type of segmentation in the pallidum that is supposed to be present in the striatum.

One must be careful in the interpretation of a case such as was reported by Wilson, in which death occurred at the age of 88. Unless serial sections were made of the basal ganglia region and stained by such accepted methods as the

Nissl on alcohol fixed material, I should not be satisfied with the statement that "the left corpus striatum was intact by microscopic examination." It seems most unusual to me that at 88 there were no small areas of softening, not only in the corpus striatum but throughout the entire brain; any conclusion drawn from such a case is apt to be misleading. One of the main features in the case that I have just reported was the fact that the patient died at the age of 25, and no account had to be taken for accessory lesions due to vessel changes.

TWO CASES SHOWING A DIAGNOSTIC TRIAD FOR THE RECOGNITION OF INCIPIENT PARKINSONISM. DR. A. M. ORNSTEEN.

This article will be published in full.

DISCUSSION

DR. W. B. CADWALADER: The signs that Dr. Ornsteen has demonstrated are of great importance in the recognition of the milder types and the early stage of the parkinsonian syndrome.

The patient he exhibited, presenting tonic spasms of the eyelids, has interested me especially, because, in 1914 (*J. A. M. A.* **63**:1380 [Oct. 17] 1914) I reported and presented before this Society two patients under the title of "progressive lenticular degeneration." One had tonic spasms of the eyelids, but, in addition to the other symptoms, there was pigmentation of the skin, associated with tremor and muscular rigidities. I called attention at that time to the resemblance this case bore to the pseudosclerosis of Westphal and Strümpell. Both eyes would be closed in tonic spasm and could not be opened voluntarily. When, however, the patient placed his finger on the orbital ridge and pushed upward, both lids could be elevated and the eyes then remained open. This appears to have been the first case in which tonic spasmodic closure of the lids was recorded. In the light of more recent experiences, it is likely that this case might now be regarded as one of chronic encephalitis.

TWO CASES OF DISEASE OF THE BASAL GANGLIA. DR. C. S. POTTS.

a. Huntington's Chorea.—M. P., a woman, aged 47, was admitted in February, 1927, to the Philadelphia General Hospital, on the service of Dr. C. S. Potts, with the history of the onset of tremors at the age of 18 years following an attack of rheumatism. The family history showed that three brothers had the same neurologic condition. Two members of the family were normal.

Physical examination disclosed fibroid pulmonary tuberculosis. There was also hypertrophy of the sternocleidomastoid muscles.

The neurologic examination showed: cranial nerves normal; reflexes normal; abdominal reflexes present; no abnormal reflexes. The gait was normal except for difficulty in the use of the right leg due to a previous fracture. The strength of the extremities was good. There was no rigidity. The head was in constant motion; this was a vertical movement. There were occasional clonic movements of the muscles of the left angle of the mouth. The head was pulled backward. There was a constant coarse tremor of the hands and arms which ceased when the arms were moved. The finger-to-nose test was difficult to perform owing to the tremor. The tremors ceased during sleep and were increased by excitement. Speech was at times explosive, due probably to tremor of the lips. Sensation was normal.

Examination of the eyes showed no pathologic condition of the fundus. A Kaiser-Fleischer ring was not present. (It was not present in any of the brothers.) The serology was normal. During the past year the patient had been losing strength. She had difficulty in getting food to the mouth because of tremor. Jerking movements of the head were more pronounced than they were a year before presentation. The chin pointed to the left. The muscles of the neck were in a state of spasm. The speech was a little slurring at times.

b. Familial Tremor.—W. M., aged 42, a white man, was admitted to the Philadelphia General Hospital in 1921, on the service of Dr. Potts. The condition began at the age of 30 with involuntary movements of the shoulders and hands; the movements increased on excitement. There was a gradual increase in symptoms until all of the body was involved. In 1920, the patient noticed that he was beginning to feel weak. He also began to have difficulty in walking. His father was said to have had "St. Vitus' dance." There was no history of other similar cases in the family.

Neurologic examination on admission (1921) showed: station normal; cranial nerves normal except for hippus; reflexes prompt and equal on both sides; no abnormal reflexes; no changes in sensation.

In 1923, the patient began to show behavior changes. He was noisy and quarrelsome. In 1925, the movements increased so that the patient could no longer work. The speech became blurred and difficult to understand. Mentally he remained normal. During 1926, there was rapid mental and physical deterioration. In 1927, the pupils were almost fixed, but the eyegrounds showed nothing pathologic. Mentally, he had further deteriorated. The serology was normal.

DISCUSSION

DR. W. B. CADWALADER: The patient just shown by Dr. Potts is a sister of the patient in the other case (case 2) of progressive lenticular degeneration that I reported in 1914 in the paper to which I have just referred. My patient had violent involuntary movements, muscular rigidity, contractures and dysarthria. He was confined to bed, or a wheel chair. The resemblance of this case to those described by Wilson in his original report was striking, and I think it was characteristic of progressive lenticular degeneration.

The patient whom Dr. Potts has just shown does not seem to me to present a characteristic case of this condition, for she does not have the same characteristic involuntary movements, nor the muscular rigidity. Her disability appears to be more in the nature of tremor.

It is of great interest to realize that in a familial disease, such as this must be, different members of the family may present different clinical pictures. Dr. Potts' case might be regarded as one of pseudosclerosis, or possibly one of chronic encephalitis, whereas, the symptoms her brother showed were much more severe and different in character. Another brother is said to have been affected.

DR. C. S. POTTS: This patient has three brothers with somewhat similar symptoms. I have seen two of them. The movements are more violent. One of them is unable to walk at all. None of them have ever had symptoms of liver disease, though, of course, the involvement of the liver is often found only at autopsy. I do not think the symptoms are all due to encephalitis. It is not likely that they all could have had it. I think that it is familial lenticular degeneration of some sort. The duration of the illness is of some interest. Usually patients with Wilson's disease live only four or five years. This patient's symptoms began at 18 and she is now 47. The brothers are older. Their symptoms began at about the same age. They have no mental disturbance. The Kaiser-Fleischer corneal ring is absent.

TWO CASES OF CHORDOTOMY, INDICATING THE DISTINCT SEPARATION OF PAIN AND TEMPERATURE FIBERS IN THE ANTEROLATERAL ASPECT OF THE SPINAL CORD, AS WELL AS THE RELATIVE POSITION OF THESE FIBERS SUPPLYING THE TRUNK AND LOWER EXTREMITY. DR. GEORGE WILSON and DR. TEMPLE FAY.

In two recent cases of chordotomy for relief of pain, it was possible with the patient under local anesthesia to section the anterolateral columns of the spinal cord, making sensory examinations while the actual sectioning of these pathways was being carried out, to guide in the proper selection of the fibers supplying the part involved.

Approximately eighteen years ago (1911), Dr. Spiller requested and had performed the first chordotomy on the human being, with section of the anterolateral columns. The cutting of the columns was undertaken for Dr. Spiller by Dr. Edward Martin in a case of intractable pain from malignancy of the sacral cord and vertebrae (*J. A. M. A.* **58**:1489 [May 18] 1912).

Since Dr. Spiller's report on the symptoms associated with the anterolateral column in 1905, he had been considering section of these fibers for relief from pain in the lower segments of the body. Formerly, rhizotomy had been resorted to. Schuller had also suggested section of the anterolateral columns for gastric crises in place of rhizotomy, which had been performed before that time. However, Dr. Spiller's case is the first one on record in which anterolateral chordotomy was accomplished in the human being and successfully demonstrated on the patient. Dr. Spiller deserves the credit for the original idea and the initial introduction of this procedure.

At the same time, Cadwalader and Sweet reported the results of anterolateral section of the cord on two dogs, with loss of pain and temperature sense following their operation, as well as some ataxia.

After these reports of 1912, chordotomy was practiced in various clinics, Foerster performing the operation in 1913. The technic of chordotomy was improved by Dr. Frazier, and the results were reported in 1920. Dr. Spiller and Dr. Frazier again reported results in 1923. Since that time a series of cases have been reported, especially from the clinic of Dr. Peet.

Recently, Foerster has demonstrated that the fibers for the sacral distribution and lower extremity are peripheral to those of the more proximal segments of the thigh and abdomen. He also pointed out the dissociation of pain and temperature sense within the spinal cord.

The following two cases represent (1) the ability under local anesthesia to select the pain fibers from the temperature fibers in the anterolateral column, and (2) the situation of these fibers within the anterolateral tract, showing the most peripheral fibers supplying the most distal segments.

CASE 1.—A patient of Dr. Wilson, a man, aged 44, complained of severe pain in the right hip. Standing and walking increased the pain to such a degree that he became unable to work. The pain radiated from the right hip to the outer aspect of the right thigh. During study at the Episcopal Hospital, roentgen examinations reported by Dr. Bromer revealed that the patient was suffering from an advanced, hypertrophic osteo-arthritis of the lumbar vertebrae. This was thought to be the cause of the pain which had incapacitated him to such an extent that he could no longer continue to work. A chordotomy was undertaken for relief from the pain.

On Nov. 27, 1928, a laminectomy was performed by one of us (Fay) and the cord exposed at the fifth thoracic segment. As the pain was unilateral and confined to the right hip and thigh, a left unilateral chordotomy was performed.

The operation was done with the patient under local anesthesia, and after careful preparation of the field, sensory examinations were made by one of us (Wilson), so that it was possible to determine accurate responses from the patient as to pain, heat, cold, tactile and deep pressure sense, just prior to introducing the knife. Having established the cooperation of the patient and the normal response in the extremities, a slight, superficial section was made along the outer aspect of the anterolateral surface of the spinal cord. This was accomplished by a sharp, triangular knife. Strangely enough, the patient did not complain of any referred pain at the moment of section, but it was immediately evident that painful stimulation of the right foot failed to elicit the response from the patient of "sharp" (pain) obtained just a moment before. Heat, cold, deep pressure sense and tactile sense were correctly distinguished in the right foot. Pain sense alone had disappeared. The examiner (Wilson) obtained a painful response when a point above the right ankle was reached on the outer surface of the foot, sensation still being retained at the instep. A slightly deeper incision was then made into

the cord, and painful sensation to pinprick was immediately lost to above the knee. Again, heat, cold, tactile and deep pressure sense remained intact in the area anesthetic to pain. The incision in the anterolateral surface of the cord was deepened then slightly, and sensation for pain disappeared to about the level of the umbilicus. All other forms of sensation were correctly called by the patient, but he could not appreciate the difference between sharp and dull on the right leg and side of the abdomen, hip and buttocks.

In order to make sure that all the fibers had been sectioned, the incision was reentered by the knife without cutting on either side of the incisional tract; the point of the knife reaching about 2 mm. within the cord. The final sensory level determined was about the seventh thoracic segment, pain being the only sensation which was lost; at times a slight diminution for temperature sense was appreciable between the two sides when compared with each other, but at no time did he fail to distinguish correctly between heat and cold.

Sensory examination revealed almost complete loss of pain sense on the right side below the tenth thoracic segment; pinprick could be appreciated occasionally over the right leg in the region of the thigh. Heat and cold were readily distinguished, although by comparative tests there was a slight diminution of these two sensations on the right as compared to the left. A marked impairment of pain sense, amounting to total loss throughout the greater portion of the field, as compared to the appreciation of temperature, although slightly diminished, was of a striking degree, and it seemed apparent that these two sets of fibers have separate representations in the tracts of the anterolateral columns.

Case 2.—This woman, also a patient of Dr. Wilson, aged 37, suffering from tabetic crises, especially abdominal pain, was operated on under local anesthesia on Jan. 23, 1929. The patient was neurotic and difficult to manage. A sensory examination made at the time of the operation by Dr. Spiller gave the following results:

"The lower limbs and the region about the anus were tested for heat, cold and pain sensations before the operation and were found to be normal.

"When the first incision was made by Dr. Fay, which he said was superficial, pain sensation was at once lost, and pinprick was called dull as high over the posterior part of the right leg as two-thirds the distance between the heel and the knee, and over the sole of the foot; apparently, pain sensation was more distinctly lost on the inner half of the sole. Ice water in these parts was called hot, and a temperature of 138 F. was equally called hot, no distinction being made by the patient. Contact was invariably recognized promptly. (Distribution of the first and second sacral segments.)

"The second incision was considerably deeper and the area of lost sensation extended immediately over the posterior parts of the right lower limb and the outer side of the limb, so far as could be reached without moving the patient from the prone position. In these parts, pinprick was felt as dull, ice water was called hot, and hot water (from 102 to 138 F.) was called hot, but the hot water was felt much more keenly in the corresponding parts of the left lower limb. It is questionable whether the sensation was truly one of warmth, even though the patient spoke of the parts feeling the hot water as hot. These disturbances extended over the buttocks and back, as far as a line indicated at the operation, which was determined after the operation to be about 1 inch (2.5 cm.) below the umbilicus. Above this line only pinprick was used for testing, as the patient was becoming tired and it was thought that her answers for temperature might be unreliable. Pinprick was felt as sharp well up to the axilla, but it was much less definitely sharp than in a part of the left side used for comparison.

"No distinction could be made as regards the location of temperature and pain sensations in the spinal cord, for both forms of sensation were affected simultaneously by the incisions. It was hoped that a distinction could be made.

"As Foerster has emphasized that the zone immediately about the anus has a certain amount of immunity from impairment of sensation after chordotomy, this

area was carefully determined in the tests, and no difference in the reactions could be found between this zone and the adjoining part of the right buttock.

"It would be difficult to obtain a more unfavorable condition for testing sensation than in a patient during the operation of dividing the fibers of sensation, as the discomfort of the patient is great, and one does not wish to prolong the operation. I think that the statements given in the note are essentially correct. The hot water at the beginning of the tests was 138 and at the end 102 F.

"It is important to note that above the line mentioned sensation was much impaired for pinprick, but not lost. This is the condition usually seen in extramedullary tumors of the spinal cord, and indicates that the pain fibers in the spinal cord were greatly damaged by the incision, but were not entirely destroyed in function."

Although in this case it was impossible to differentiate the pain and temperature pathways, due primarily to a faulty cutting edge of the knife, nevertheless, this case, like case 1, illustrates the situation of the fibers for pain and temperature regarding their distribution to the trunk and lower extremity; the most superficial fibers in the anterolateral columns of the cord supply the sacral portion of the skin surfaces. The lower extremity is represented more superficially than the trunk. As the knife is advanced in depth the anesthesia rises, so that it was possible in this case, introducing the knife at about the fourth thoracic segment, to section even fibers supplying the sixth thoracic dermatomere.

In this case, anesthesia for pain and temperature was established on the right side (left chordotomy) to the sixth thoracic dermatomere. A more superficial section of the cord was made on the other side as this case required a bilateral chordotomy (right chordotomy) with the resultant anesthesia as high as the third lumbar dermatomere on the left.

DISCUSSION

DR. W. G. SPILLER: I believe that the pain and temperature tracts are in contact in the lateral columns of the spinal cord but that these tracts are distinct from one another. I suggested to Dr. Fay that by cutting the cord from without inward, and by limiting the incision to a small area, he might be fortunate enough to sever the pain fibers and leave the temperature fibers intact. This he has done in large measure, and so far as I know he is the first to accomplish this result. The operation of anterolateral chordotomy is capable of refinement, and we shall probably attain such perfection that with the patient under local anesthesia we may determine the depth necessary for the knife to penetrate to accomplish the loss of pain sensation to the level desired, and may impair or destroy one tract of sensory fibers and leave the adjoining tract intact or nearly intact. When the pain has a burning character (causalgia) it may be necessary to sever the temperature fibers as well as the pain fibers. It was evident that, in the patient in whom I was examining the sensory loss during the performance of chordotomy, the upper level of the anesthetic area rose as the knife penetrated deeper.

Book Reviews

EPILEPSY. Harvard Medicine Monographs, volume XIV. By WILLIAM G. LENNOX and STANLEY COBB, Harvard Medical School. Price, $3.50. Pp. 197. Baltimore: Williams and Wilkins Co., 1928.

The authors give a thoughtful presentation of the modern point of view of epilepsy. It is a summary of the work they have done personally, with a consideration of the work of others.

There are four acceptable theories for epilepsy: (1) the irritation theory, based on experiments in electrical excitation and the pathologic changes found in jacksonian epilepsy; (2) the release theory, which holds that convulsions result from the activity of the lower centers, because of suspension of the function of the higher; (3) a short circuit theory, which is allied to the release theory but is more local, and (4) the explosive theory, which holds that seizures arise as a general widespread change in the brain tissue, dependent on some sudden metabolic alteration.

The authors believe that epileptic seizures are neurologic phenomena, usually motor, caused by a sudden change in the nerve cells. The etiologic factors, which might spring the trigger of this mechanism, may be numerous and varied. They then elaborate the factors involved in these convulsions. There is first a discussion of the pathologic condition of the nerve tissue, both at clinical examination and at autopsy. Then follows a discussion of the functional abnormalities of the nerve tissue, with an elaboration of the physicochemical changes in the nerve cells, and the evidence from the effects of drugs.

From the psychogenic point of view, the abnormalities found outside of the nervous system are discussed in detail, including those of the circulatory, autonomic and respiratory systems, the gastro-intestinal tract, the endocrine glands, the blood, the spinal fluid, the urine, the feces, protein metabolism, carbohydrate metabolism, fat metabolism, acid-base and induced alkalosis and induced acidosis. Statistical considerations and practical considerations, such as examination of the patient, prophylaxis and treatment, are also discussed. A summary appears after each subject, and then there is a final summary with an extensive bibliography.

Enough has been said to indicate the scope of this book, which regards epilepsy chiefly from the physicochemical point of view. The authors consider that the recent advance in the knowledge of epilepsy demonstrates that changes in the physicochemical process in the body and presumably, therefore, in the brain may profoundly modify seizures. They point out that Hippocrates had somewhat the same point of view and that modern physicians are rapidly catching up with the views of the father of medicine.

In brief, the evidence so far gathered indicates that in certain epileptic patients subject to frequent seizures, the seizures may be precipitated by lack of oxygen and alkalosis, however induced; whereas seizures may be prevented by increased oxygen tension in the tissues and acidosis, however induced. Linked with these two factors is that of the water balance of tissues.

This work is by far the most scientific presentation of epilepsy which has appeared and is an indication that real progress is being made in the elucidation of the causes of this oldest of diseases.

ANATOMY AND THE PROBLEM OF BEHAVIOR. By G. E. COGHILL, Member of the Wistar Institute of Anatomy and Biology, Philadelphia. Price, $3.00. Pp. 110. New York: The Macmillan Company, 1929.

Professor Coghill delivered these lectures before the University of London, an honor paid to few American scientists. In itself it is an expression of the high regard in which Coghill's work is held. The problem consisted in making parallel

studies of the development of behavior and the development of the nervous system. It seemed to him "basic to a scientific study of behavior to know whether the behavior pattern of an animal develops haphazard or in an orderly manner; and that, if it should be found that behavior develops in an orderly manner, then there should be a corresponding order of development structurally and functionally in the nervous system."

This question was taken up in the first lecture. The *Amblystoma* was found to be a splendid animal for this purpose. In brief, Coghill found that "the behavior pattern develops in a regular order of sequence of movements, which is consistent with the order of development of the nervous system and its parts; (2) in a relatively precise manner physiological processes follow the order of their embryological development in the functions of aquatic and terrestrial locomotion and feeding;" (3) behavior develops from the beginning through the progressive expansion of a perfectly integrated total pattern and the individualism within it of partial patterns which acquire various degrees of discreteness.

The second lecture outlines the origin of the conducting mechanism from preneural structures and functions, and emphasizes the fact that the preneural functions overlap the neural functions, in the development of the mechanism of behavior. It is apparent from this that the conventional idea of conduction does not give a complete picture of how the nervous system plays its rôle in the development of behavior.

The last lecture takes up the question of how the preneural process of growth and differentiation in the nervous system, and particularly the individual neuron, participates in the function of the nervous system as a whole.

Regarding behavior, the author comes to the conclusion that man is more than the sum of his reflexes, instincts and immediate reactions of all sorts. He is all of these plus his creative potential for the future. The real measure of the individual, accordingly, whether lower animal or man, must include the element of growth as a creative power. Man is a mechanism, but he is a mechanism which, within his limitations of life, sensitivity and growth, is creating and operating himself.

This work is full of ideas and suggestions which can be adapted to the growth and behavior of humans, and is an indication of how much real knowledge can be acquired by such comparative studies on the lower animal.

CLINIQUE MÉDICALE DES ENFANTS. By P. NOBÉCOURT. Price, 45 francs. Pp. 374. Paris: Masson & Cie, 1928.

This is the fifth volume in the series of pediatric clinics by the same author and is devoted to a consideration of some of the more common diseases of the nervous system in children. As the title implies, the material is presented in clinical fashion, stressing particularly the history in each case, the methods employed for diagnostic purposes, the difficulties and problems encountered and the treatment instituted.

The first two chapters are devoted to a consideration of several cases of epidemic encephalitis, both typical and atypical forms, with a discussion of the etiology, cardinal symptoms, various syndromes and modes of onset. The author particularly emphasizes the occurrence of ocular palsies and somnolence in typical cases. The consideration of the sleep cycle leads to the topic of the next chapter —"The Occurrence of Somnolence in Various Affections of Childhood" — in which the author stresses the frequency of this symptom in children and indicates that it may be due to any one of a great variety of causes.

The next lesson (chapter IV) is devoted to a discussion of "Parkinson's Syndrome and Epidemic Encephalitis in Children." The material and presentation are not new, conforming to the well known descriptions of other observers.

In chapter V the author takes up the subject of typhoid fever in children, particularly with reference to the occurrence of psychic and encephalitic phenomena during the course of the disease and during convalescence.

The ensuing chapters are devoted to the following subjects: (1) "The etiology of Sydenham's Chorea," with remarks on the relation of chorea to epidemic encephalitis, in which the author maintains that Sydenham's chorea constitutes a clinical entity, caused by a neurotropic virus, perhaps related to the virus of epidemic encephalitis. (2) "The Various Types of Onset of Tuberculous Meningitis"—typhoid, apoplectic, ataxic, delirious, somnolent and convulsive—followed by a chapter on the occurrence of the disease in heredosyphilis and the differential diagnosis. (3) "Meningococcic Meningitis"—the various types and early clinical manifestations. (4) The different types of "pneumococcic meningitis." (5) "Convulsions and Congenital Syphilis." (6) "A Case of Cerebral Tumor," with an excellent discussion of the significance of headaches in children. (7) "Peripheral Facial Paralysis" and its many causes. (8) An interesting chapter on "Herpes Zoster" with an adequate summary of present knowledge of this condition. (9) "Infantile Paralysis," its onset, course and treatment. (10) "Diphtheritic Paralyses," and the last chapter, a case of "Friedreich's disease."

Each chapter is a clinical lesson, concise, to the point and enhanced by a level-headed discussion of theories and facts. The book should prove of special value to the student and practitioner, and to the pediatrician as well as to the neurologist.

SCHIZOPHRENIE-SCHIZOBULIE. By FRITZ SCHULHOF, M.D., PH.D. Price, 4 marks. Pp. 108. Vienna: Franz Deuticke, 1928.

Schulhof, in this monograph, critically reviews the symptomatology of schizophrenia in search of some possible element, or elements, validly postulable as actually basic or nuclear to the superdeveloping semeiologic structure. This, he believes, he has found in what he speaks of as the "ich-denke-gefühl," and devotes the bulk of his section on schizophrenia to the detailed development of this thesis. The "ich-denke-gefühl," following an original lead from Kant, is taken to comprise the appreciation or realization on the part of the individual that he himself is the author or source of his thinking. This personal identification with ideational content and activity is regarded as absolutely sine qua non for the mentally sound but, according to the author, is lost in the schizophrenic person and represents the primary pathologic focus from which develops, secondarily, the abundant and characteristic symptomatology usually noted clinically. In the heboid type, this loss is accepted as such, without affect arousal and without especial effort as to explanation or adjustment, psychotic or otherwise. In the elaborative forms, paranoid and hallucinatory, these special manifestations are construable, largely, as attempts in interpretation and harmonization of the subjective difficulty thus occasioned.

An additional basic factor is also brought forward, the so termed "ich-will-gefühl." This is described as the personal sense of volitional effectiveness and capacity, or "power," and is present in all normal persons. When this sense is lost, the author thinks that a special pathologic condition may result. This state, which he designates "schizobulia," may lead to active psychotic or dereistic elaboration in the process of satisfactory personal explanation and adaptation. Schizophrenia and "schizobulia" are closely related apparently, with both fundamental pathologic qualities often present in the same patient, although instances do seem to occur in which only one factor is overly manifest. Schulhof's point, it seems to the reviewer, is well taken and is worthy of really serious thought.

ZUR PATHOPHYSIOLOGIE DER "AKUTEN HALLUZINOSE" WERNICKE'S UND DES MELANCHOLISCHEN SYMPTOMENKOMPLEXES. By FRITZ HARTMANN, aus der Universitäts-Nervenklinik zu Graz. Studien zur Klärung der Pathophysiologie der psychopathologischen Symptomenkomplexe. Price, 4 marks. Pp. 68. Berlin: S. Karger, 1928.

On the ground of clinical symptoms of pulmonary fibrosis in one or both apexes, confirmed by roentgen examination, a positive reaction to tuberculin, positive

Abderhalden reactions to lung and brain and an increase of alkali reserve, Hartmann finds himself justified in framing a physiopathologic theory of hallucinosis. Similarly, he speculates elaborately on the explanation of melancholia on the grounds of an Abderhalden reaction to liver. The logical structure of the detailed speculations may satisfy certain minds. The references to the vegetative system and to the balance between certain compounds (particularly calcium-potassium) of body fluids are well used, and the presumptive disorders in both the vegetative system and the humoral state give, to superficial reading, a satisfactory explanation of respiratory and mental disturbances and of the interrelation between them.

Unfortunately for the theory, pulmonary changes on a tuberculosis basis of one or both apexes are commonplace and therefore it is easy to find them in any disease, and not less frequently in normal persons than in the conditions discussed by Hartmann.

The physiopathologic study is based on "objective" observations, but these observations, because of their ubiquity, have hardly more significance than the equally ubiquitous carious teeth or any other chronic infection. The author does not consider the question of focal infection in his study, but his physiologizing argument should interest those who are preoccupied with the urge to construct elaborate reasoning when one needs facts that can speak more convincingly for themselves and that deal with what constitutes the case in which the "process" appears pertinent, and in which it is a mere incident or is present without any specific effects.

A History of Pathology. By Esmond R. Long. Price, $5. Pp. 290, with illustrations. Baltimore: Williams & Wilkins Company, 1928.

Fielding H. Garrison states in the preface to this work: "Dr. Long has arranged this survey in twelve compact chapters, illustrating the different new departures in the progress of pathology." While the book is intended primarily for the medical student, it is excellent not only for the pathologist but for everyone interested in the history of medicine, for the development of pathology goes hand in hand with progress in medicine. The book is most readable. The twelve chapters are arranged as follows: 1. The Pathology of Antiquity. 2. Galen and the Middle Ages. 3. The Pathology of the Renaissance. 4. The Seventeenth Century. 5. Morgagni and the Eighteenth Century. 6. The Paris School at the Opening of the Nineteenth Century. 7. Pathology in England in the First Half of the Nineteenth Century. 8. Rokitansky and the New Vienna School. 9. Virchow and the Cellular Pathology. 10. Pathological History and the Last Third of the Nineteenth Century. 11. The Rise of Bacteriology and Immunology. 12. Experimental and Chemical Pathology.

As one can see from a perusal of the contents, the material is taken up in sequence, and in a way in which even one who does not usually care for the ordinary medical histories not only can read and understand all the subjects but can actually enjoy them. The illustrations are well chosen. The reviewer agrees thoroughly with Garrison when he says: "This book can be cordially recommended to any physician who wishes to know the origins and evolution of the basic discipline of scientific medicine." Dr. Long is to be heartily congratulated on his book.

Sex Development in Apes. By Harold C. Bingham. Price, $2.50. Pp. 165 Baltimore: Johns Hopkins Press, 1928.

A full account is here rendered respecting sex development and behavior in certain monkeys and apes, chiefly chimpanzees. This report, which is most informative, is based not only on cross-sectional observation but also on extremely painstaking, long-section ontogenetic studies.

Of particular significance appears to be the length of time required for the establishment of sex maturity and the associated adult copulation pattern. This involves a number of years, at least, and is marked by considerable preliminary

experimental trial and error, especially in relation to the determination of a final and mechanically effective coital formula. Much of the same, it is felt, represents exploratory procedure necessitated by lack of facility for expression in terms of language. In this connection, it seems that sight and touch are the sense mediums most involved. During the formative period, the author finds that sex response organization involves the following phases: sucking, clinging, nuzzling, genital contact, embracing, cuddling, exploring and erection. In addition, and as a forerunner to actual copulation, certain special manifestations are found to be of definite importance: manipulation, playful aggression, attack, flight, retreat, evasion, tantrums, ornamentation and coquetry. Especially interesting and illuminating are Bingham's observations relative to homosexual, masturbatory and exhibitionistic manifestations, which seem not uncommon, also respecting sex arousal as induced through the presence or interest of human beings.

HIRNPATHOLOGISCHE BEITRÄGE. DR. KARL SCHAFFER, Budapest. Volume 7. Pp. 516. Budapest, 1928.

The seventh volume of Hirnpathologische Beiträge is dedicated to Dr. Karl Schaffer, director of the psychiatric Neurological Clinic and Histological Institute of Budapest, on the occasion of the completion of forty years of work. There is a foreword by S. Ramón y Cajal, and then there is a brief summary of Professor Schaffer's work with a bibliography containing a list of 108 papers that have appeared in the general literature and 35 papers that have appeared only in Hungarian. Besides, six volumes of these "Arbeiten" have appeared during the incumbency of Professor Schaffer. A perusal of this list gives an index of the splendid work that has come out of the clinic and laboratory of Professor Schaffer not only from himself but from the workers who have been trained by him.

The seventh volume consists of a series of eighteen papers from the Institute. One of these, at least, has appeared as an original in the ARCHIVES OF NEUROLOGY AND PSYCHIATRY. The others have been published elsewhere.

AIDS TO PSYCHOLOGY. By JOHN H. EWEN. Price, $1.50. Pp. 166. New York: William Wood & Company, 1929.

In this book, the author gives a compilation of the main facts of psychology. The book is intended to offer a means of rapid revision following a study of the larger textbooks on the subject. The aim throughout is to emphasize those points useful to students studying for a diploma in psychologic medicine. For its purpose, the book is adequate.

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